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ABSTRACT

Intended for child health care providers, the text contains information on improving preventive efforts in nutrition, particularly those focused on prevention of the major health problems which are nutrition related (obesity, atherosclerosis, dental caries, and anemia). Part I focuses on screening of individual children likely to be at risk of nutritional disorders and offers suggestions for followup in the form of further studies or treatment or both. Topics covered in Part I include the following: information about the community; information about the family and child; questionnaires about diet and eating habits for infants, preschool and young school-age children, and school-age children and adolescents; physical findings; dental inspection; height and weight; head circumference; skinfold thickness; laboratory studies; and roentgenograms. Part II presents discussions of obesity, atherosclerosis, dental caries, and iron-deficiency anemia prevention. The text includes graphs, sample questionnaires, tables, and illustrations. (SBH)

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Nutritional Disorders of Children

Prevention, Screening, and Followup

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FOREWORD

In both health planning and health care delivery there is increasing recognition that only by preventing the occurrence of disease can one hope to achieve any significant improvement in the Nation's health. Preventive health care in childhood promotes the development of children to their fullest potential by influencing life-long attitudes and practices in nutrition, providing for the early detection of nutritional problems as well as intervention and followup, and reducing disability and dependency from illness. The improvement of child health and nutrition in the United States is one of the major concerns of the Bureau of Community Health Services.

This publication is addressed to all providers of health care for children—private physicians, hospital pediatric units, school health programs, child care institutions, and others in the community involved in child health service. It was prepared to assist them in improving preventive efforts in nutrition, particularly those focused on prevention of the major health problems which are nutrition-related—obesity, atherosclerosis, dental caries, and anemia. Specific guidance and recommendations about methodology and standards to use for a minimal assessment, for a more extensive diagnosis, and for followup of a child's nutritional status are also included.

Planners and providers of child health care will find the guidance helpful as a basis for establishing standards, policies, and procedures for nutritional assessment in health care delivery systems. It should also be useful in identifying and strengthening the data base which is essential for planning and evaluating nutrition services, in developing a collaborative approach to nutrition problems among health personnel, and in improving the quality and continuity of nutrition services delivered to children in community health programs.

The publication was prepared by Samuel J. Fomon, M.D., in his capacity as medical expert in nutrition for the Bureau of Community Health Services. Dr. Fomon, Professor of Pediatrics at the University of Iowa, Iowa City, has worked in the field of clinical nutrition for many years and is widely recognized as an authority in pediatric nutrition. His coauthors—Thomas A. Anderson, Ph.D., Stephen H. Y. Wei, D.D.S., and Ekhard E. Ziegler, M.D.—are faculty members of the University of Iowa.

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PREFACE

This publication has evolved from a 10-year effort to present guides on screening for nutritional status and prevention of iron-deficiency anemia in child health programs.

In 1966 the Children's Bureau published "Suggested Guidelines for Evaluation of Nutritional Status of Preschool Children."* In 1970 the Maternal and Child Health Service (which had previously been a part of the Children's Bureau) assembled a group of consultants to revise these guidelines. The revision, "Screening Children for Nutritional Status: Suggestions for Child Health Programs," (Maternal and Child Health Service, 1971) restricted consideration to children less than 10 years of age and was concerned with screening of groups of children rather than an assessment of nutritional status of individuals. Because iron-deficiency anemia was recognized as a major nutritional problem among infants and small children, "Prevention of Iron-Deficiency Anemia in Infants and Children of Preschool Age" (Fomon, 1970) was published by the Maternal and Child Health Service.

The present publication, while drawing from the content of the previous publications, differs in scope and purpose. It concerns children from birth through adolescence. As described in Part I, the screening process is directed at identification of individual children likely to be at risk of nutritional disorders. For children so identified, suggestions are made for followup in the form of further studies or treatment or both.

A preliminary draft of Part I was reviewed by a group of experts in various aspects of nutritional assessment at a meeting in Rockville, Maryland, on June 10, 1975. On February 11, 1976, a second group of consultants, primarily representing potential users of the publication, reviewed the penultimate draft, including Part II.

Part II concerns prevention of four important nutritional disorders that may occur or have their origin in childhood: obesity, atherosclerosis, dental caries, and iron-deficiency anemia.

Although it presents model questionnaires, this publication is not a manual for nutrition surveys. Rather, it is meant to offer assistance to those involved in delivery of health care within a community. The presentation concerns screening measures directed to children seeking and receiving care through such community programs as maternal and child health services, crippled children services, migrant health projects,

* References appear at the end of Part I (p. 53).

Appalachia projects, community health centers, health maintenance organizations, National Health Service Corps sites, EPSDT (Early and Periodic Screening, Diagnosis, and Treatment) programs, and other health programs involved in screening and followup. It is not aimed at screening all children in the community.**

I should like to acknowledge specific help from a number of persons. Mary C. Egan of the Bureau of Community Health Services recognized the need for the publication and offered suggestions and criticisms throughout its preparation. Becky Huff Epes of the C&Y project at the University of Virginia Medical School aided in the development of the Appendix to Part I. Dr. Peter V. V. Hamill of the National Center for Health Statistics provided preliminary charts and descriptions to permit development of the text relative to Figures 4–11. Dr. Alvin M. Mauer of the Committee on Nutrition of the Mother and Preschool Child, National Academy of Science—National Research Council consulted with the authors about iron-deficiency anemia. Many other individuals read sections of the text and offered valuable suggestions, including the consultants listed on pages 122 and 123.

SAMUEL J. FOMON, M.D.
Iowa City, Iowa
1976

** It is evident that a satisfactory survey of the population under 18 years of age would require a sophisticated sampling design; such design is beyond the scope of this publication.

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PART I
SCREENING AND FOLLOWUP

In planning health care delivery for infants and children [1] it is particularly important to identify the nature and extent of common health problems, including nutritional problems. Assessment of nutritional status is assumed to represent only one aspect of total assessment of health. Knowledge of the frequency and severity of nutritional problems in children presenting themselves for care will permit reasonable allocation of resources for solving the more important nutritional problems, thereby contributing to improved health within the community, and will provide a basis for program evaluation. Without such knowledge, significant nutritional disorders may be ignored or an unwarranted investment of funds and effort may be made in combatting an imagined or trivial problem. For this reason, some screening for nutritional disorders should be carried out in every health care delivery system.

A successful effort in screening and followup of health problems will depend to a large extent on the understanding and motivation of health personnel. The need for and importance of screening must be understood. The reasons that specific procedures and methods have been selected should be explained in detail to the personnel. Proper application of the methods and interpretation of results require thorough understanding. Motivation for precise recording of results is unlikely to come about unless the importance of the data has been discussed. Health care personnel should receive orientation regarding all procedures and methods, including those that do not relate directly to their own assignments. In this way, each member of the health care team will have a broader view of the entire approach and collaborative working arrangements will be facilitated.

Although the primary purpose of screening is the identification of individuals at nutritional risk, assessment of nutritional status of those seeking care will also be of enormous value in providing data regarding nutritional disorders prevalent in the community, in indicating changes with time in prevalence of disorders affecting those receiving care and, especially, in identifying *individuals* with nutritional disorders so that

[1] Throughout this publication infants are considered to be individuals less than one year of age and children, unless otherwise specified, are all other individuals who have not yet reached 18 years of age.

intervention measures may be initiated. Such information is useful in program planning and evaluation.

Even a minimal screening program will require knowledge of the community and of food intakes and physical findings of the children. At least a few laboratory analyses will be necessary. Under some circumstances, a more elaborate (and also more expensive and time-consuming) approach to evaluation of nutritional status will be feasible and desirable. Suggestions are presented both for a minimal and for a more extensive approach to nutritional status screening and followup.

Information About the Community

Planning a screening program aimed at identifying individuals likely to be at nutritional risk will be greatly aided by knowledge of the demographic characteristics of the community in which the health care delivery system operates and by information about the food and water supply. A number of valuable suggestions for obtaining relevant information about the community are included in the chapter Community Assessment of Nutritional Status of the symposium report, *Nutritional Assessment in Health Programs* (Christakis, 1973). In many instances, some or most of the desired information about the community can be obtained from census data, from health, welfare or social service agencies, from a city planning group, housing authority, the school board, the chamber of commerce or other community agencies.

Certain information usually available from the local health department will contribute insights into the probable health status of the community. Such information includes percentage of infants with birth weights less than 2500 g, percentage of births to mothers less than 16 years of age, neonatal and infant mortality figures, and a listing of the most prevalent infectious diseases. Local hospitals will usually have data on most common reasons for admission. Data on income by census tract, extent of unemployment, and educational status of the community are also likely to be correlated with the frequency of nutritional disorders.

As stated in the publication previously cited (Christakis, 1973), the nutrition services available in the community through public and private organizations should be evaluated. For each agency it is desirable to inquire whether nutritionists are available to agency staffs, whether effective infant and child health programs are in operation, and whether these include a mechanism for nutritional evaluation. It is necessary to determine whether maternity clinics are adequate and whether they include sound nutrition programs. The availability of public programs directed toward coronary heart disease, diabetes, obesity, hypertension, alcoholism and drug addiction should be explored and the nature of their nutrition component should be ascertained.

The availability of various food assistance activities in the community should be determined. These include the food stamp program, feeding in day care centers, Headstart programs, preschool and school feeding, and food assistance programs for pregnant women, infants and children. The extent of participation in such programs should be determined.

Although local or regional factors sometimes transcend ethnic considerations, feeding practices and choices of foods frequently differ remarkably from one ethnic group to another and therefore the racial and ethnic composition of the community should be known. Information regarding shopping practices, including relative frequency of patronage of supermarkets, small grocery stores, and health food stores may provide valuable insights. The number and extent of patronage of stall vendors and snack stands should be determined, with particular attention to those located near schools. It is also important to determine whether food prices increase at the time of issuance of welfare checks and whether ease of check cashing encourages individuals to patronize smaller stores with higher prices.

It is desirable to determine the attitudes of community leaders toward nutritional problems in the community. Of particular significance in this regard are administrators (mayor, city council, etc.), county medical societies and other influential local groups.

Knowledge of the composition and community consumption patterns of various staple foods may be of considerable value. Most states have laws requiring enrichment of commercially prepared breads and rolls with iron, riboflavin, thiamin, and niacin. Enrichment of these foods with calcium and vitamin D is optional.[2] In communities where rice, corn (hominy) grits or tortillas are a dietary staple, it is important to determine whether products fortified with iron and vitamins are available. Particularly in areas in which goiter is found in children, knowledge of the availability and usage of iodized salt is valuable.

Although most fluid whole milk sold by dairies is fortified with vitamin D, fluid whole milk not fortified with vitamin D is available in rural areas and in many cities, often at a slightly reduced price. Whether or not such milk is readily available within a community should be determined and an estimate made of the extent of its use; if possible, it should be determined whether such use is general or is restricted to specific subgroups. Most fluid low-fat milks are fortified with vitamins A and D. On the other hand, powdered skim milk may not be so fortified.

The major source or sources of drinking water (e.g., community water supply, individual wells) should be determined. Information

[2] Information about laws relating to flours and grains in individual states is available from the American Institute of Baking, 400 East Ontario Street, Chicago, Illinois 60611.

about fluoride content of water from these sources is usually available from State or local health authorities.

Information About the Family and the Child

The frequency of nutritional deficiency disorders is likely to be correlated with the economic status of the family (Abraham et al., 1974; Owen et al., 1974). An index of the economic status of the family is therefore useful. Among the simplest indices of economic status is a comparison of actual income with the most recent United States Department of Agriculture estimated cost of feeding a low income family (Orshansky, 1968).[3] However, family income needs to be interpreted in light of the participation of any family member in a food assistance program such as school feeding or supplemental food program for needy mothers, infants and children. Other assistance, such as medical care, should also be noted.

Information about family housing can provide important information regarding social and environmental deprivation. Knowledge of mean occupancy per room (e.g., 0.5 to more than 2.0), water supply, sanitation facilities and adequacy of the kitchen should be determined. The latter is of particular importance because of its influence on the ability to store and prepare food.

Information relating to a child's past medical history and current state of health will not always be available in the child's health records and may need to be obtained separately. Among the various items that may be useful to record are birth weight, past history of serious or chronic illness and presence of any current illness.

General information about the individual child is often important in assessing his nutritional status. Motor development of an infant will ordinarily be retarded in the case of severe or prolonged illness. The age at which a child begins to walk represents a developmental landmark that will be recalled by most parents of preschool children. This landmark is useful in assessing progress during infancy.

Retarded growth, the most common manifestation of gross nutritional deficiency, may reflect serious illness of non-nutritional origin. Proper interpretation is therefore impossible unless a record is made of each serious or prolonged illness. This knowledge of past or current illnesses may also aid in interpreting other manifestations suggesting nutritional deficiency.

As in all aspects of screening and followup, eliciting adequate information about the family and child is a collaborative effort in which the

[3] A widely used index of socioeconomic status (as opposed to economic status alone) consists of a number derived from a weighting of values assigned to an individual's education, occupation and income (Hollingshead, 1957).

activities of pediatrician, nutritionist, nurse and social worker must be well coordinated.

Questions About Diet and Eating Habits

The questions that follow are aimed at identifying at least some of the children who may be at nutritional risk because of unusual eating habits or monotonous or unusual diets. The questions are designed to select from a larger group of children those few (perhaps 5 or 10%) receiving a diet most likely to be deficient or excessive in one or more nutrients and in need of further dietary evaluation or counseling. These children would then be seen by a qualified nutritionist. *It is important to note that the questions are not meant to determine the child's recent intake of calories, protein, iron or specific vitamins.*

It is preferable for the questions to be asked verbally and the answers recorded by a sympathetic individual who has been instructed in technics of eliciting frank replies. Although it is not necessary for the individual to have had extensive training in nutrition or dietetics, training in asking the questions and recording the responses is essential. Such training will logically be provided by the nutritionist.

The nutritionist will review all completed questionnaires and decide which children are likely to profit most from an interview with a nutritionist. Frequently, it will be desirable for the nutritionist to consult with the physician in charge. In some instances of unusual diets, it may be important for the physician, nurse or social worker to conduct a more extensive interview with the child and/or his caretaker. The questionnaires that follow are applicable to various age groups: Questionnaire I—infants less than one year of age; Questionnaire II—preschool children and young school-age children whose parent or caretaker will answer for them; Questionnaire III—older school-age children and adolescents who are able to answer for themselves. Comments on interpretation are presented on page 12.

QUESTIONNAIRE I
INFANTS
(FROM BIRTH TO AGE 1 YEAR)

	Yes	No
Is the baby breastfed?	☐	☐
If yes, does he also receive milk or formula?	☐	☐
If yes, what kind? _____		
Does the baby receive formula?	☐	☐
If yes, ready-to-feed ☐		
concentrated liquid ☐		
other ☐		
How is formula prepared (especially dilution)? _____		
Is the formula iron-fortified?	☐	☐
Does the baby drink milk?	☐	☐
If yes, whole milk ☐		
2% milk ☐		
skim milk ☐		
other ☐		
Specify _____		
How many times does he eat each day, including milk or formula? _____		
Does the baby usually take a bottle to bed?	☐	☐
If yes, what is usually in the bottle? _____		
If the baby drinks milk or formula, what is the usual amount in a day?		
less than 16 oz ☐		
16 to 32 oz ☐		
more than 32 oz ☐		

Please indicate which, if any, of these foods the baby eats and how often.

	never or hardly ever (less than once a week)	sometimes (not daily but at least once a week)	every day or nearly every day
Eggs	☐	☐	☐
Dried beans or peas	☐	☐	☐
Meat, fish, poultry	☐	☐	☐
Bread, rice, pasta, grits, cereal, tortillas, potatoes	☐	☐	☐
Fruits or fruit juices	☐	☐	☐
Vegetables	☐	☐	☐

	Yes	No
If the baby eats fruits or drinks fruit juices every day or nearly every day, which ones does he eat or drink most often? (Not more than three).		
_____		
_____		
_____		
If the baby eats vegetables every day or nearly every day, which ones does he eat most often? (Not more than three).		
_____		
_____		
_____		
Does the person who cares for the baby have use of a		
working stove?	☐	☐
refrigerator?	☐	☐
piped water?	☐	☐
Does the baby take vitamin or iron drops?	☐	☐
If yes, how often? _____		
what kind? _____		
Is the baby on a special diet now?	☐	☐
If yes, what is the reason?		
allergy; specify type of diet ☐		
_____		
weight reduction; specify type of diet ☐		
_____		
other; specify type of diet ☐		
_____		
Who recommended the diet? _____		
Does the baby eat clay, paint chips, dirt or anything		
else that is not usually considered food?	☐	☐
If yes, what? _____		
how often? _____		
Do you think the child has a feeding problem?	☐	☐
If yes, describe _____		
_____		

QUESTIONNAIRE II

PRESCHOOL CHILDREN AND YOUNG SCHOOL-AGE CHILDREN WHOSE PARENT OR CARETAKER WILL ANSWER FOR THEM

	Yes	No
Does the child drink milk?	☐	☐
If yes, whole milk ☐		
2% milk ☐		
skim milk ☐		
other ☐		
Specify _____		
If yes, how much?		
less than 8 oz ☐		
8-32 oz ☐		
more than 32 oz ☐		
(For children less than 4 years of age.)		
Does the child drink anything from a bottle?	☐	☐
If yes, milk ☐		
other ☐		
Specify _____		
Does the child take a bottle to bed?	☐	☐
If yes, what is usually in the bottle?		

How many times a day does the child usually eat (including snacks)? _____

Please indicate which, if any, of these foods the child eats and how often.

	never or hardly ever (less than once a week)	sometimes (not daily but at least once a week)	every day or nearly every day
Cheese, yogurt, ice cream	☐	☐	☐
Eggs	☐	☐	☐
Dried beans, peas, peanut butter	☐	☐	☐
Meat, fish, poultry	☐	☐	☐
Bread, rice, pasta, grits, cereal, tortillas, potatoes	☐	☐	☐
Fruits or fruit juices	☐	☐	☐
Vegetables	☐	☐	☐

If the child eats fruits or drinks fruit juices every day or nearly every day, which does he eat or drink most often? (Not more than three).

_____

_____

_____

	Yes	No
If the child eats vegetables every day or nearly every day, which ones does he eat most often? (Not more than three).		
_____		
_____		
_____		
Does the child usually eat between meals?	☐	☐
If yes, name the 2 or 3 snacks (including bedtime snacks) that the child has most often.		
_____		
_____		
_____		
Does the person who cares for the child have use of a working stove?	☐	☐
refrigerator?	☐	☐
piped water?	☐	☐
Does the child take vitamin or iron drops or tablets?	☐	☐
If yes, how often? _____		
what kind? _____		
Is the child now getting a special diet?	☐	☐
If yes, what is the reason?		
allergy; specify type of diet ☐		
_____		
weight reduction; specify type of diet ☐		
_____		
other; specify reason for diet and type of diet ☐		
_____		
Who recommended the diet? _____		
Does the child eat clay, paint chips, dirt or anything else not usually considered food?	☐	☐
If yes, what? _____		
how often? _____		
How would you describe the child's appetite?		
good ☐		
fair ☐		
poor ☐		
other (specify) ☐		
_____		

QUESTIONNAIRE III

SCHOOL-AGE CHILDREN AND ADOLESCENTS WHO ANSWER THE QUESTIONS THEMSELVES

	Yes	No
Do you drink milk?	☐	☐
If yes, whole milk ☐		
2% milk ☐		
skim milk ☐		
other ☐		
Specify _____		
_____		

Please indicate which of the following foods you eat and how often.

	never or hardly ever (less than once a week)	sometimes (not daily but at least once a week)	every day or nearly every day
Cheese, yogurt, ice cream	☐	☐	☐
Eggs	☐	☐	☐
Dried beans, peas, peanut butter	☐	☐	☐
Meat, fish, poultry	☐	☐	☐
Bread, rice, pasta, grits, cereal, tortillas, potatoes	☐	☐	☐
Fruits or fruit juices	☐	☐	☐
Vegetables	☐	☐	☐

If you eat fruits or drink fruit juices every day or nearly every day, which ones do you eat or drink most often? (Not more than three).

_____

_____

_____

If you eat vegetables every day or nearly every day, which ones do you eat most often? (Not more than three).

_____

_____

_____

Do you usually eat anything between meals?	☐	☐
If yes, name the 2 or 3 snacks (including bedtime snacks) that you have most often.		
_____		
_____		
_____		
Do you or the person who prepares your meals have use of a working stove?	☐	☐
refrigerator?	☐	☐
piped water?	☐	☐

	Yes	No
Do you take vitamins or iron?	☐	☐
If yes, how often? _____		
what kind? _____		
Are you on a special diet?	☐	☐
If yes, what is the reason?		
allergy; specify type of diet ☐		
_____		
weight reduction; specify type of diet ☐		
_____		
other; specify reason for diet and type of diet ☐		
_____		
Who recommended the diet? _____		
Do you eat clay, paint chips or anything else not usually considered food?	☐	☐
If yes, what? _____		
how often? _____		

Interpretation

It is hoped that these few simple questions will aid in selecting from the larger group of children a small subsample particularly deserving of more extensive review of food intake or of particular laboratory studies. The nutritionist will need to spend a few minutes studying each completed questionnaire to determine which sets of responses are likely to indicate nutritional risk.

In the case of infants (Questionnaire I), the questions are directed particularly at detecting major errors in feeding: too great dilution or too great caloric concentration of a formula, a calorically inadequate diet because skim milk is being fed, hazard from nursing bottle caries (p. 83), an inadequate or excessive quantity of milk or formula consumed (less than 16 oz or more than 32 oz of milk or formula), complete avoidance of certain food categories (e.g., an older infant who never or hardly ever eats any fruits or vegetables), inadequate facilities for food preparation and/or storage, excessive intake of fat-soluble vitamins, adherence to a special restrictive diet (especially if not under a physician's supervision), pica.

In questioning preschool children and young school-age children whose parent or caretaker will answer for them, the questions (Questionnaire II) must be individualized to some extent in relation to the age and developmental stage of the child. For young preschool children, many of the questions designed for infants will be applicable. For older preschool children and young school-age children, the questions will be similar to those in Questionnaire III.

In the case of older children and adolescents, the major aim of the dietary questions (Questionnaire III) is the identification of individuals with bizarre or monotonous diets, in the belief that such individuals are likely to be at relatively high nutritional risk. When certain major categories of foods are never or hardly ever eaten, when meals are rarely eaten and snacking is common, when cooking and storage facilities are inadequate, or when the child is receiving a restrictive diet, an interview with a nutritionist will be indicated.

Each nutritionist will have a personal approach to eliciting more detailed information. One approach is indicated in the Appendix (p. 56).

Physical Findings

Physical examination, including dental examination, is essential in nutritional screening. Special attention should be paid to such general features as pallor, apathy and irritability. The skin should be examined for petechiae, ecchymoses or dermatitis, and the skeletal system for cranial bossing, enlarged joints or costochondral beading. The lower extremities should be examined for edema, and visible thyroid enlargement should be recorded. The presence of heart murmurs or of

enlargement of liver or spleen may suggest presence of a chronic disease responsible for growth retardation.

A physical finding suggesting nutritional abnormality should be looked upon as a clue rather than as a diagnosis. Costochondral beading should not be interpreted as evidence of rickets without roentgenographic confirmation. Thyroid enlargement should not be interpreted as evidence of iodine deficiency without appropriate laboratory confirmation.

McGanity (1970) has offered some insight into the reliability of specific physical signs of nutritional deficiency. In Panama in 1967 two well trained individuals examined each of 895 adult subjects. Presence or absence of abnormal hair, filiform papillary atrophy, follicular hyperkeratosis, swollen red gums, angular lesions, glossitis and goiter was noted by each examiner. With respect to subjects classified as having abnormal hair, agreement between the two examiners occurred in only 31 percent. Among individuals identified by one of the examiners as having filiform papillary atrophy, agreement between examiners occurred in only 50 percent. Agreement between examiners with respect to other physical signs was generally not much better. Thus, the detection of clinical signs of deficiency in the hair, eyes, tongue, skin and nails will rarely be of much value.

Physical findings will be meaningful primarily in identifying diseases that may interfere with growth and general health. While such disorders (e.g., congenital heart disease, chronic liver disease) may lead to unsatisfactory nutritional status, they must be distinguished from nutritional deficiencies resulting from failure to provide an individual with food of adequate quantity and nutritional quality.

Screening Followup

Any potentially important abnormality detected by physical examination will require further confirmation and/or interpretation through biochemical, roentgenographic or other methods. When the finding pertains to the general evaluation of health (e.g., a heart murmur, an enlarged and tender liver), followup studies are particularly important. The nature and medical significance of a heart murmur should be determined, with referral to a cardiologist if necessary. Liver function studies may be necessary in evaluation of liver enlargement.

Dental Inspection

As discussed in Part II (Chapter 3), dental caries is widespread among all age groups and can be prevented, at least to some extent, through good nutritional practices. A critical aspect of the physical examination therefore is the examination of the teeth and supporting structures.

Ideally, the inspection should be carried out by a dentist or experienced dental hygienist with adequate physical facilities (i.e., dental chair, adequate lighting, full range of instruments). In many instances, this will not be possible because neither such highly qualified personnel nor fully adequate facilities will be available. However, a reasonably adequate dental screening can be made even in rather poor surroundings by other health personnel. For example, a dental assistant equipped with penlight and suitable diagnostic instruments for intraoral use can be trained to examine and chart dental caries.

Screening Followup

In most centers delivering health care, dental caries will be so prevalent that adequate followup will present a major problem. Emergency care for the relief of pain and treatment of acute oral infections should be given first priority. Next, the children with most extensive oral and dental disease without pain should be treated. When these groups have been cared for, particular attention should be directed toward the youngest children (those one to three years of age). Older age groups can be added as resources become available. For all children, especially the youngest, the dental inspector should provide advice about oral hygiene and the nutritionist, in collaboration with the dental consultant, should advise about decreasing the frequency of exposure of the teeth to refined carbohydrates and about the desirability of providing fluoride supplements and/or topical applications of fluoride (p. 87).

Height (or Length) and Weight

Height (or length) and weight are among the most important indices of nutritional status and *should be accurately determined*. The metric system is preferred for recording the data.

Weight

Body weight should be measured with appropriate-sized beam scales with nondetachable weights. Two sets of scales are desirable, one for infants and small children that permits reading to the nearest 10 g and another set for older children (and adults) that permits reading to the nearest 20 g.[4] Scales should be calibrated at 3 to 4 month intervals.[5]

[4] Suitable scales may be obtained from the Toledo Scales Company, Toledo, Ohio 43612, from the Douglas Homs Company, Inc., Burlingame, California 94010, or from a local dealer in weights and measures. A portable scale of 50 kg capacity is available from the Douglas Homs Company, Inc. (model 51 KGT 1111); a portable scale of 100 kg capacity is also available from this source.

[5] A local dealer in weights and measures will sometimes be willing to carry out the calibration. When this cannot be arranged, standard weights (one 5 kg and several 10 kg weights) may be obtained from the Toledo Scales Company or from the Douglas Homs Company, Inc.

Measurements of weight of infants are preferably made with subjects unclothed. Young children should be unclothed or clothed only in undergarments. Adolescents should wear undergarments and every effort should be made to protect them against embarrassment.

Length or Height

During the first two years of life, length (i.e., with body supine) rather than height (i.e., standing) should be measured.

LENGTH:

Measurement of length is difficult and should not be attempted unless satisfactory equipment and two trained examiners are available. In many instances, it will not be practical to measure length routinely.

One person holds the infant's head with the head positioned so that the line of sight is directly upward and applies gentle traction to bring the crown of the head into contact with the fixed headboard. A second trained person (not the mother) holds the infant's feet, knees and hips completely extended, toes pointing directly upward and, also applying gentle traction, brings the movable footboard to rest firmly against the infant's heels (Fig. 1). An examining table, simply and inexpensively modified as described by Falkner (1961), will provide a suitable measuring apparatus. Alternatively, a portable measuring board of the type shown in Figure 2 can be utilized.

HEIGHT:

Children 24 months of age and older may be measured in the standing position. However, two examiners and much patience are usually needed to obtain standing measurements of children less than 3 years of age. It is strongly recommended that platform scales with movable measuring rods not be used for measuring height. Satisfactory equipment can be provided by fixing a measuring stick or tape to a true vertical flat surface, either a wall or a rigid, free-standing measuring device. The child should stand on a horizontal bare floor or platform with his bare heels together, back as straight as possible with the heels, buttocks, shoulders and head touching the wall or vertical surface of the measuring device; the line of sight should be horizontal. A block, squared at right angles against the wall, is then brought to the crown of the head and the measurement noted (Fig. 3).

Description of Reference Data

Figures 4–11 present data on size of American children as compiled by a task force of the National Center for Health Statistics (Hamill et al., 1976a,b)* in accordance with recommendations of the Committee

*Charts may be obtained from Ross Laboratories, Columbus, Ohio 43216.

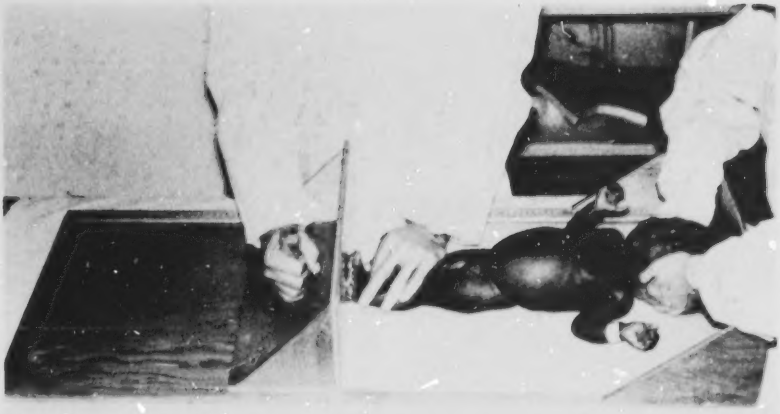

Figure 1 Technic of measuring recumbent length (Falkner, 1961).

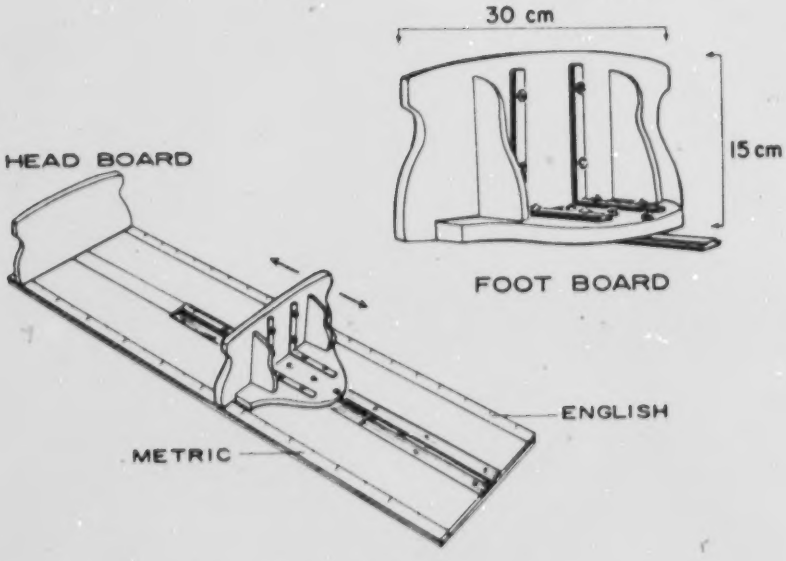

Figure 2 Apparatus used for measuring length (Fomon, 1974).

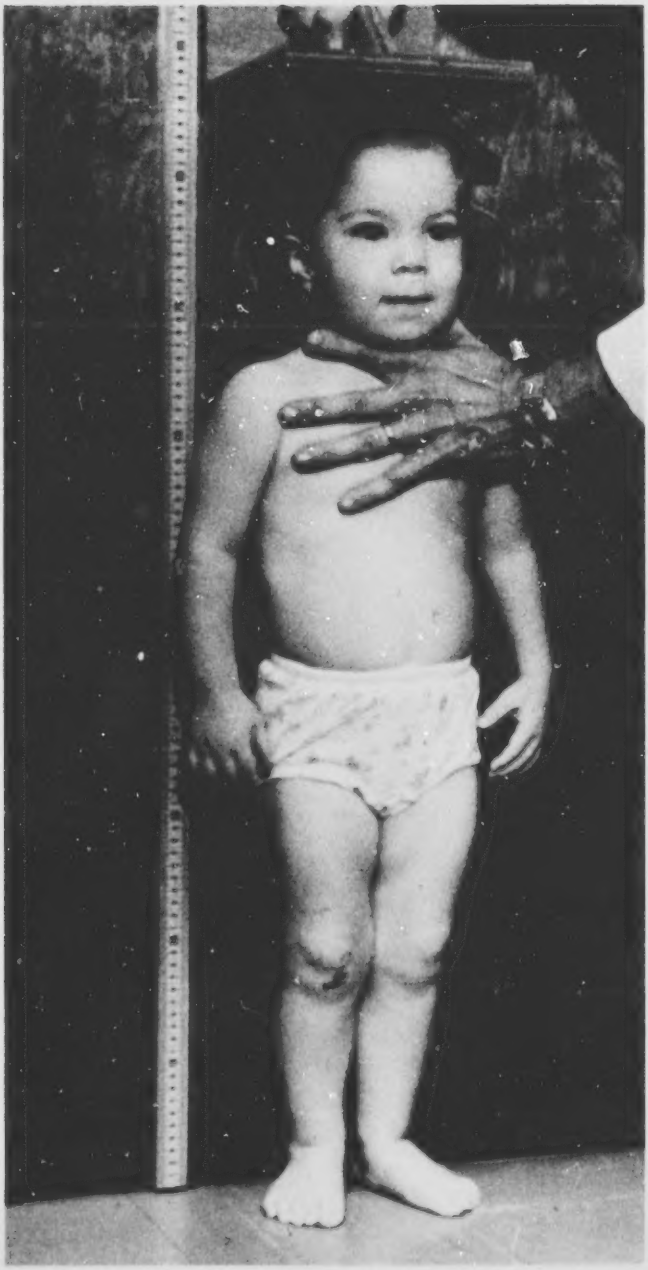

Figure 3 Technic for measuring height (Fomon, 1974).

on Nutrition Advisory to CDC (1974). One set of the charts (Fig. 4–7) presents data from the Fels Research Institute concerning 867 children followed longitudinally from birth, the first child born in 1929 and the last child in 1970. Although every child was not measured at every age, 700 to 800 children were measured at each of the following ages: birth, 1 month, 2 months, 6 months; 9 months, 1 year, 1½ years, 2 years, 2½ years and 3 years. Multiple births were purposely over-represented in the sample. In preparing Figures 4–7, data on four sets of triplets were excluded but data on 14 sets of twins were included. Forty children (4.6%) weighed less than 2500 g at birth. The charts include weight for age and length for age (Fig. 4 and 5) and weight for recumbent length (Fig. 6 and 7).

The set of charts for children 2 through 17 years of age (Fig. 8–11) was constructed from data of the National Center for Health Statistics from three cycles of surveys: Health Examination Survey (HES) Cycle II, 7119 children 6 through 11 years of age (1962–65) representative of 12.1 million males and 11.7 million females in this age group in the United States; HES Cycle III, 6768 individuals 12 through 17 years of age (1966–70) representative of 11.5 million males and 11.2 million females in this age group; Health and Nutrition Examination Survey (HANES), children 2 through 17 years of age (1971–74). In this latter survey, 14,000 individuals (of all ages) were measured, including 2342 children 2 through 5 years of age. Data on 4183 children 6 through 17 years of age supplemented the data from the two cycles of the Health Examination Survey. These are entirely cross-sectional data, no child contributing measurement data on more than one occasion. Because of the similarity and efficiency of the stratified probability sample designs, the data from the three National Center for Health Statistics surveys could be combined.

Weight for age and height for age are presented in Figure 8 (for boys) and in Figure 9 (for girls). Weight for height for prepubescent children is presented in Figure 10 for males and in Figure 11 for females. For reasons described by Hamill et al. (1976a), for purposes of these charts prepubescent males were defined as those less than 11.5 years of age with height less than 145 cm; prepubescent females were defined as those less than 10 years of age with height less than 137 cm.

Each figure includes the 5th, 10th, 25th, 50th, 75th, 90th and 95th percentile values. The method of smoothing the data to obtain the various percentiles has been described (Hamill et al., 1976b).

Interpretation: Small Size for Age or Low Weight for Height

Deficiency of total caloric intake, of protein, or of any other essential nutrient may be responsible for decreased rate of growth and, ultimately, for abnormally low height (or length) or weight. In countries in which

protein-calorie malnutrition is common, children with least weight for age, height (or length) for age, or weight for height demonstrate a greater prevalence of nutritional abnormalities than do other children. Although a similar relation between small size and prevalence of nutritional abnormalities has not been demonstrated in the United States, it does seem possible that the smallest children are deserving of closer scrutiny than others.

The aim of nutritional screening of children in the United States is identification of children with mild or marginal nutritional abnormality. The simple index of weight for age, so widely used in countries where severe protein-calorie malnutrition is prevalent, is unlikely to have the sensitivity desirable for use in the United States. A low value of height for age raises the possibility of relatively long-term illness or nutritional deficiency. On the other hand, when height for age is above the 10th percentile but weight for height is less than the 5th percentile, one may suspect the presence of acute or subacute illness or nutritional deficiency. It is evident that any illness of sufficient severity to cause weight loss will result in rapid decrease in the ratio of weight to height.

When measurements of height (or length) and weight have been made before the time of the current evaluation, they should be analyzed to determine past rate of growth. When feasible, the child should be followed with sequential measurements so that rate of growth (more sensitive than size in detecting abnormalities) may be recorded.

Screening Followup: Small Size for Age or Low Weight for Height

Steps to be taken for further evaluation of the children so identified will vary somewhat on the basis of differences in local resources. A more comprehensive assessment of food consumption should be obtained (see Appendix, p. 56). In all instances, it will be desirable to review the medical, social and family history in detail. Often it will be desirable for the physician, nutritionist, social worker and other health care personnel to discuss the available information and to develop a suitable strategy for proceeding. If a child has severe congenital heart disease or cystic fibrosis of the pancreas, the approach to undernutrition will be different from that of a child whose family is unable to provide food or provides food that does not meet requirements for energy or essential nutrients. Lack of basic nutrition information will require a different approach than the one needed for inadequate cooking facilities or income.

In addition to the physical examination desirable for all children, simple laboratory studies (hemoglobin concentration, hematocrit, leukocyte count, erythrocyte sedimentation rate, urinalysis) and roentgenograms of the chest should be performed. When it can be arranged, a member of the health team should visit the home to observe the

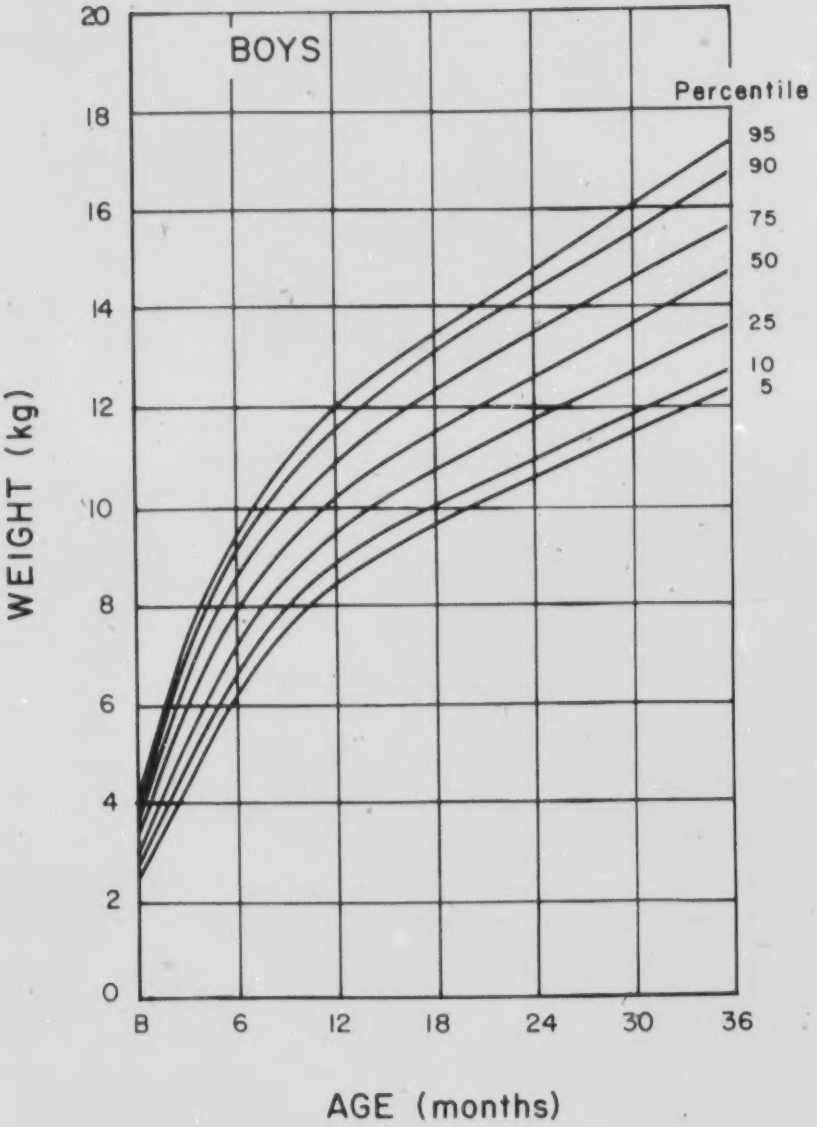

Figure 4a Weight for age of boys from birth to 36 months (Hamill et al., 1976a).

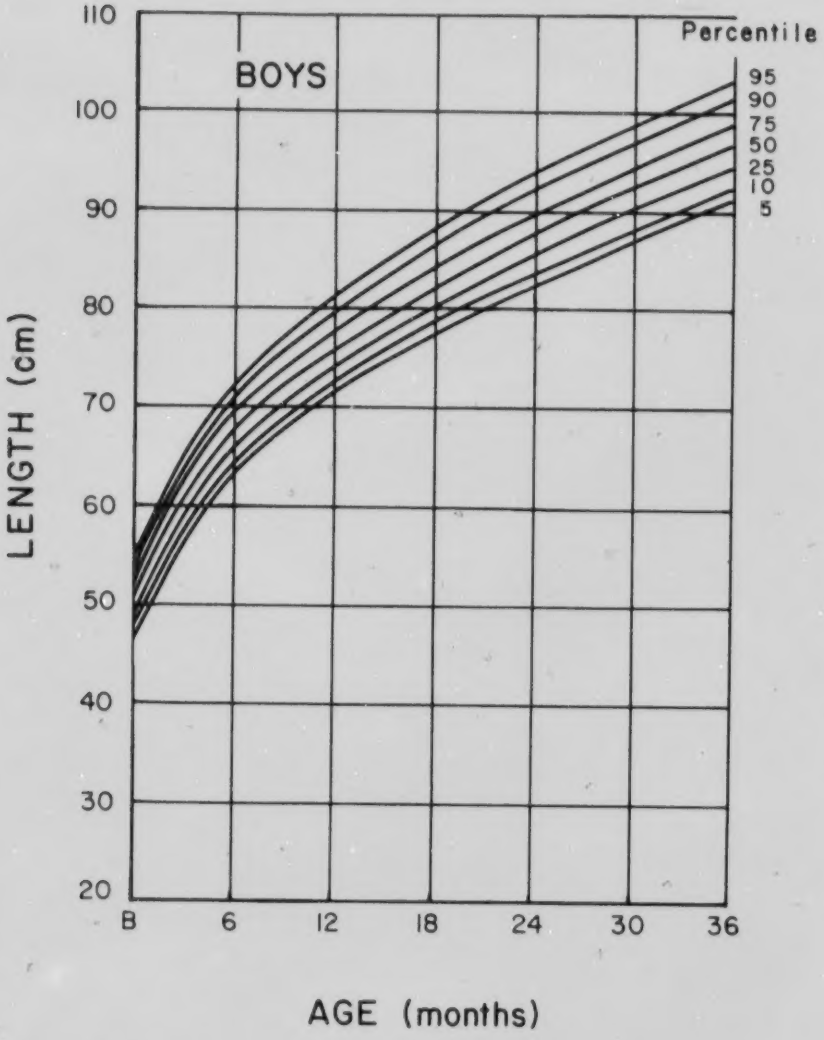

Figure 4b Length for age of boys from birth to 36 months (Hamill et al., 1976a).

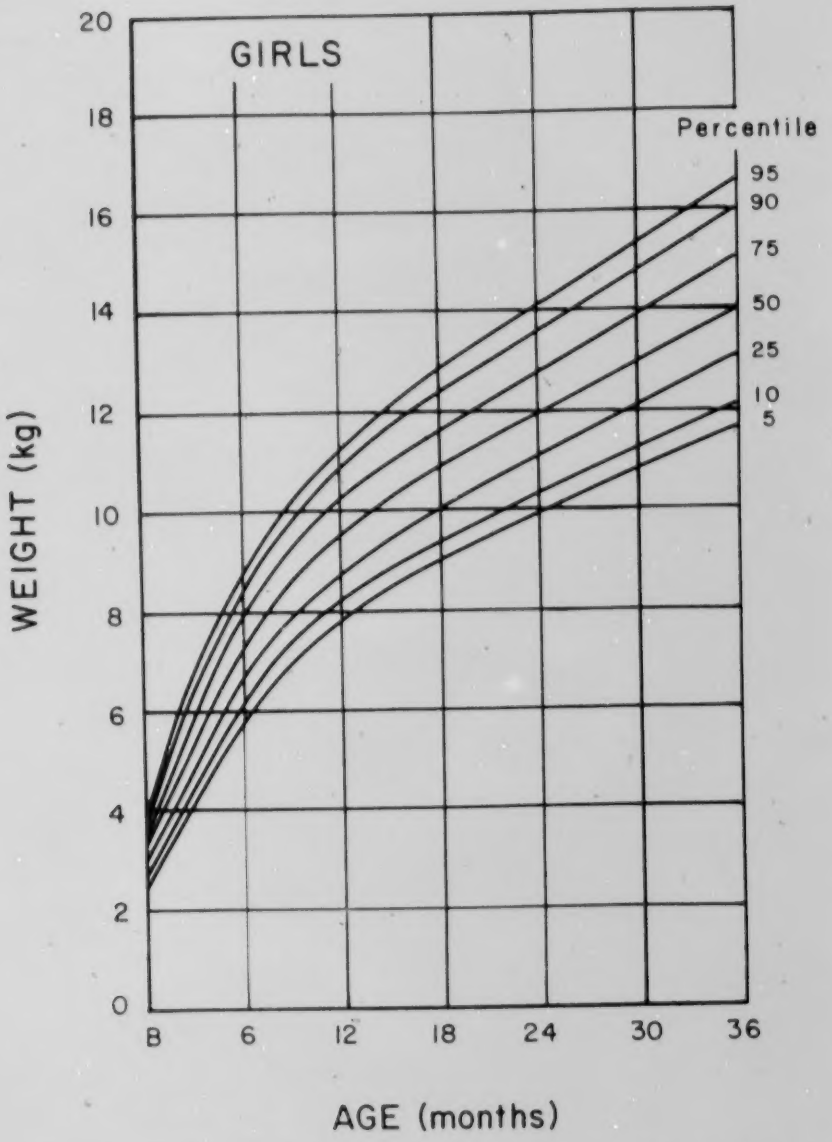

Figure 5a Weight for age of girls from birth to 36 months (Hamill et al., 1976a).

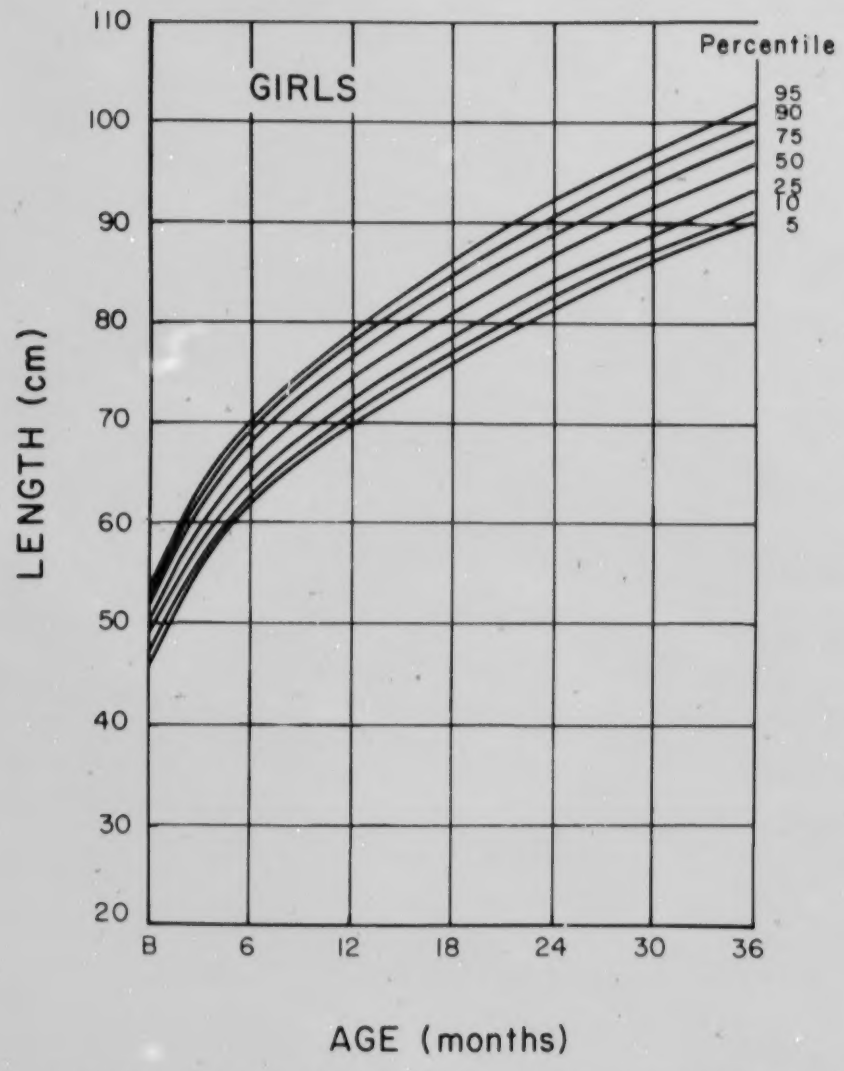

Figure 5b Length for age of girls from birth to 36 months (Hamill et al., 1976a).

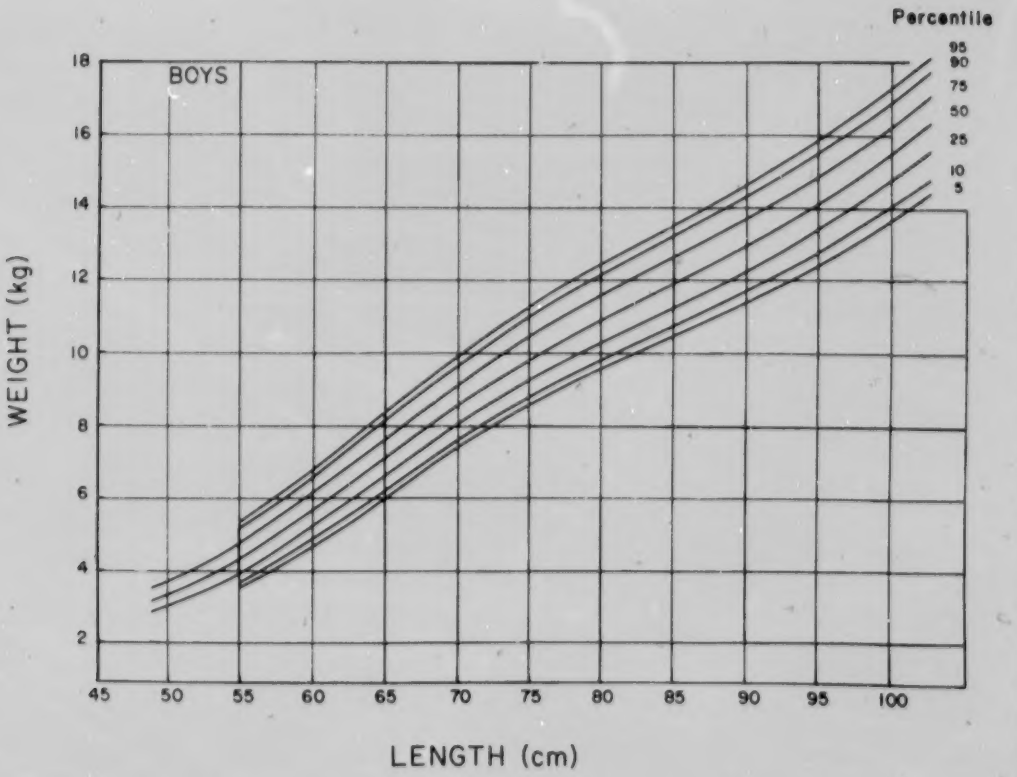

Figure 6 Weight for length of boys from birth to 36 months (modified slightly from Hamill et al., 1976a).

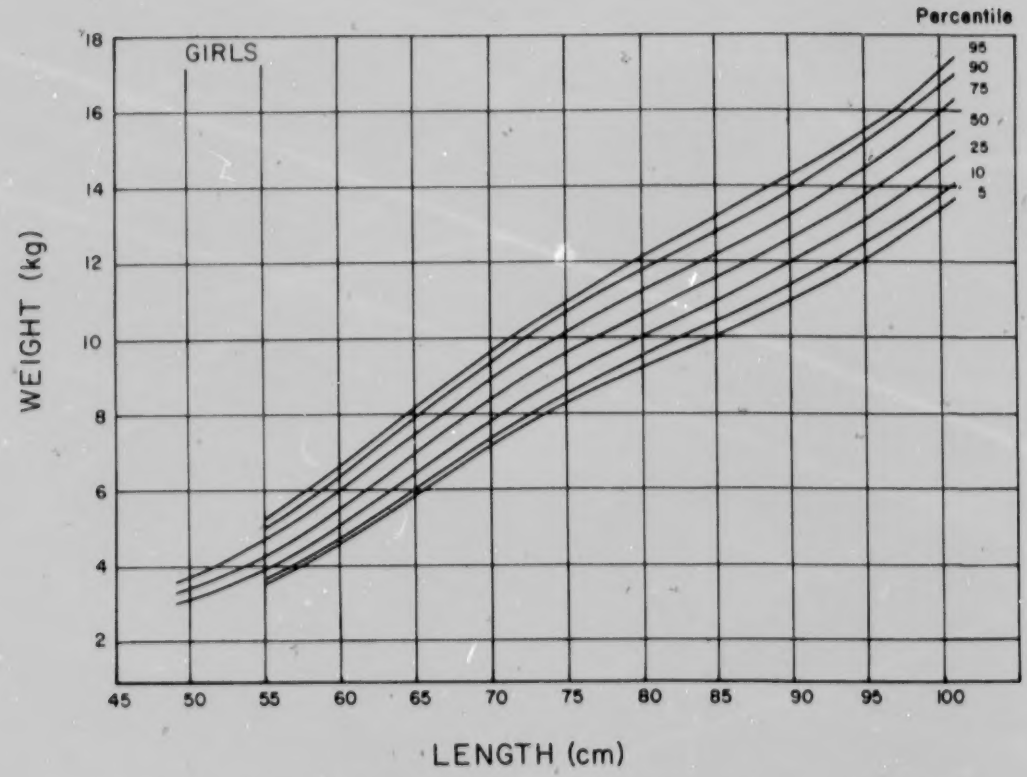

Figure 7 Weight for length of girls from birth to 36 months (modified slightly from Hamill et al., 1976a).

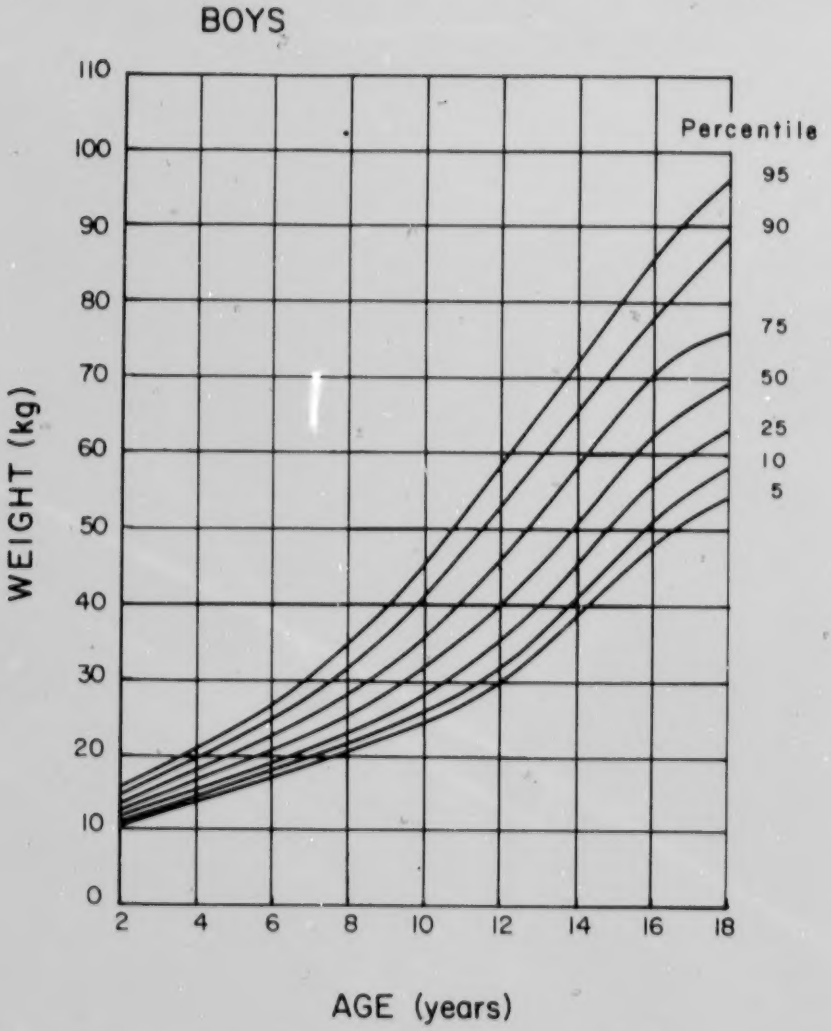

Figure 8a Weight for age of boys from 2 to 18 years (Hamill et al., 1976a).

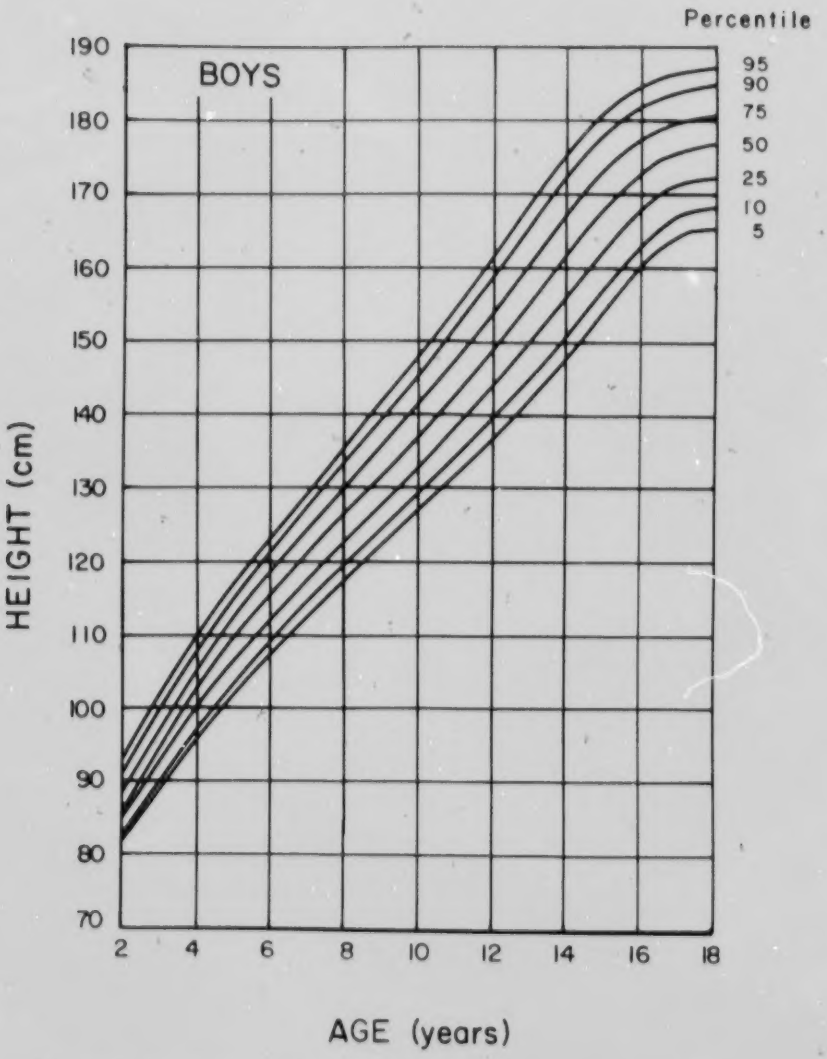

Figure 8b Height for age of boys from 2 to 18 years (Hamill et al., 1976a).

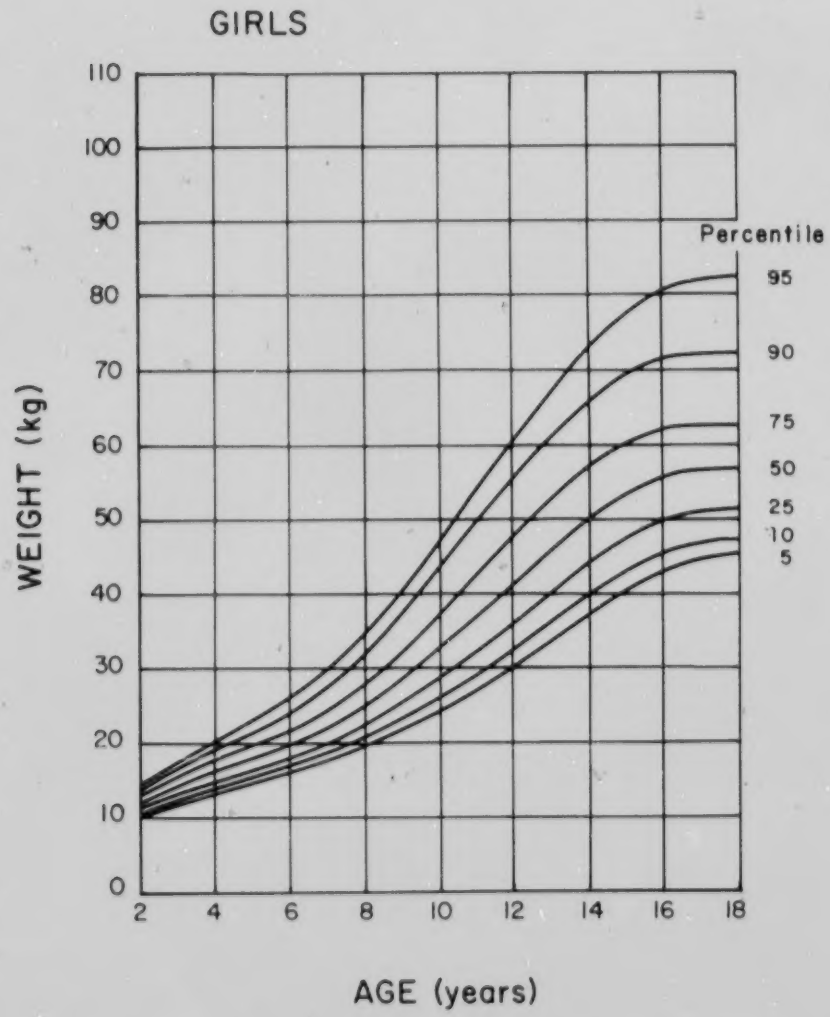

Figure 9a Weight for age of girls from 2 to 18 years (Hamill et al., 1976a)

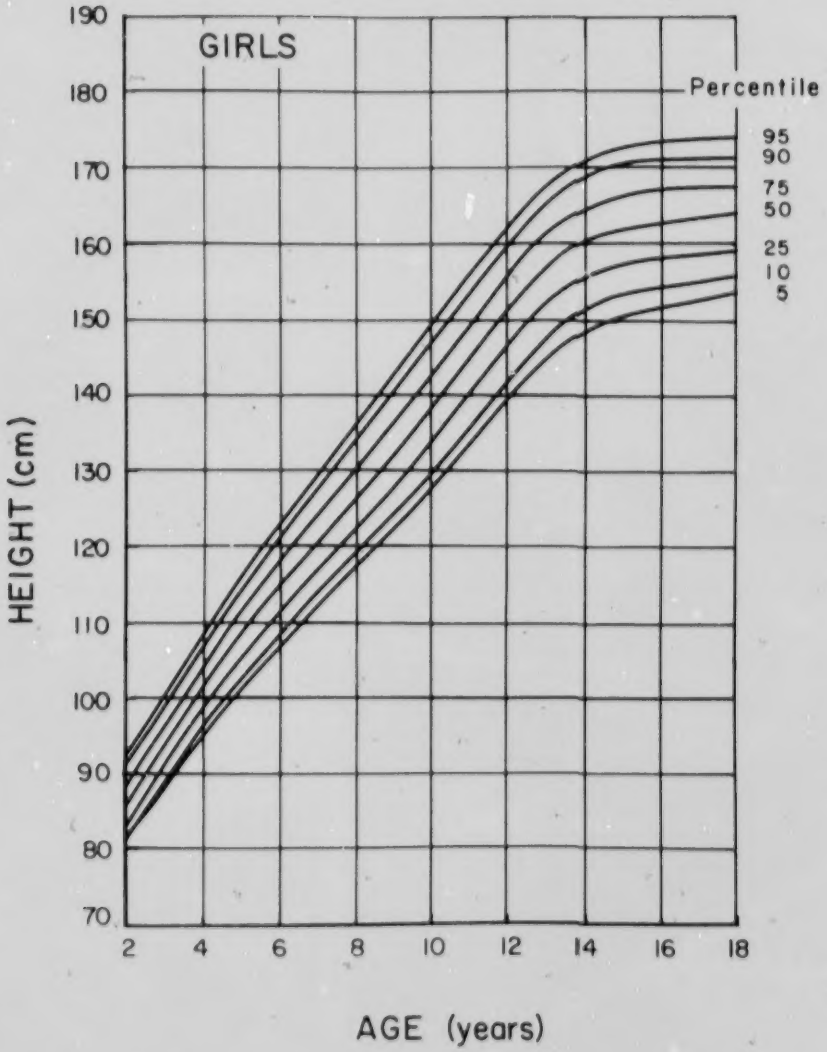

Figure 9b Height for age of girls from 2 to 18 years (Hamill et al., 1976a).

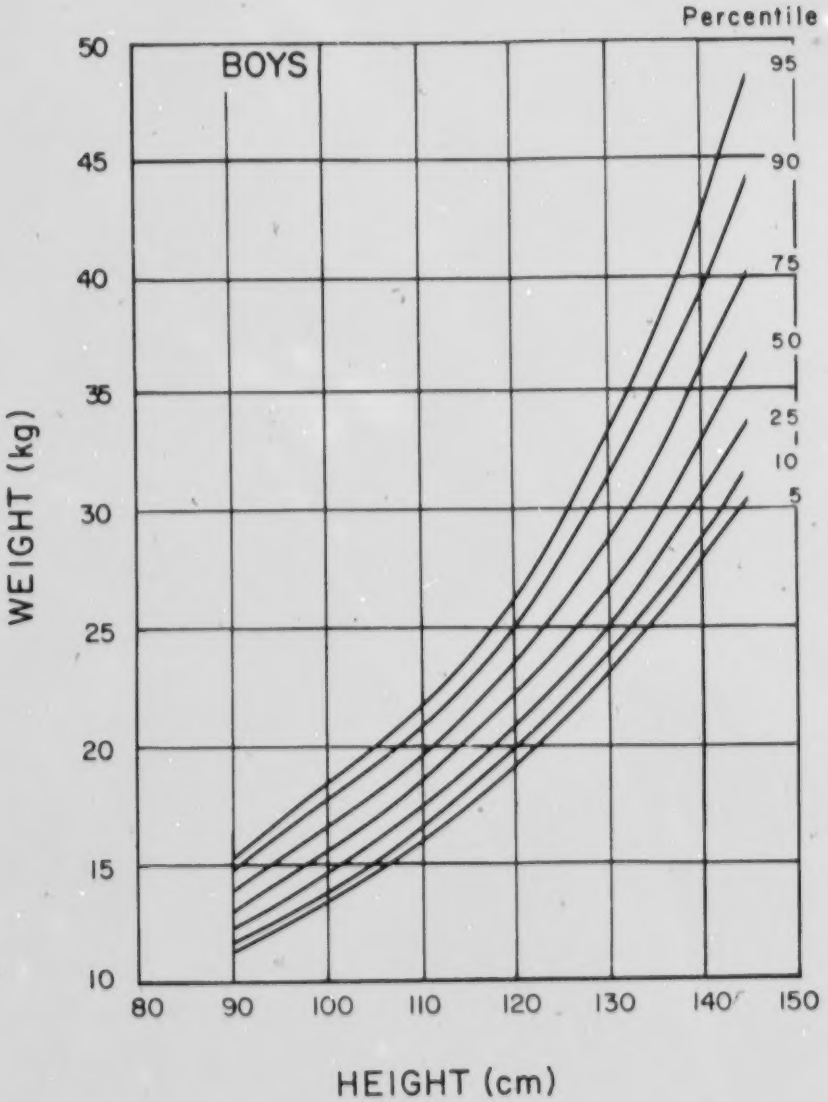

Figure 10. Weight for height of prepubertal boys (Hamill et al., 1976a). This chart is applicable to boys less than 11.5 years of age with height less than 145 cm.

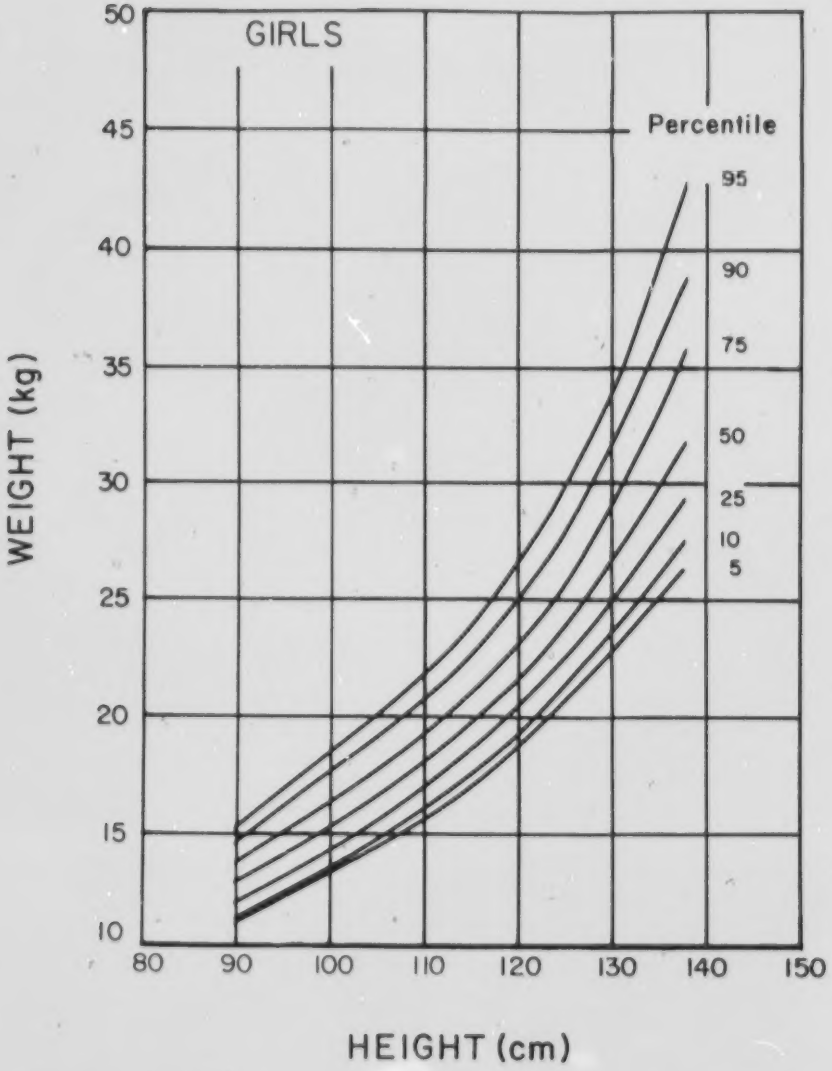

Figure 11 Weight for height of prepubertal girls (Hamill et al., 1976a). This chart is applicable to girls less than 10.0 years of age with height less than 137 cm.

condition of the home, the availability of appropriate food, and methods of food preparation and feeding.

In some communities, the number of children with height (or length) for age or with weight for height (or length) less than the 5th percentile of the reference data will be too large to make it feasible to carry out the studies listed. Under such circumstances, further studies should be directed to the children with least height (or length) for age and with least weight for height (or length).

Interpretation: Excess Weight for Height

As will be discussed in Part II, Chapter 1, of this publication, a satisfactory definition of obesity will presumably be based on the percentage of body weight accounted for by fat. Skinfold thickness is likely to be correlated in a general way with body fat content but it will frequently not be feasible to measure skinfold thickness in routine screening programs. When measurements of skinfold thickness cannot be made, weight for height (or length), or even weight for age, interpreted in the light of height (or length) for age, may be useful. For children less than 9 years of age, satisfactory reference data on weight for height (or length) are available (Fig. 6, 7, 10, 11). Weight for height (or length) greater than the 95th percentile may be considered presumptive evidence of obesity. For children over 9 years of age, the use of weight alone to indicate obesity is unsatisfactory (except in extreme instances of obesity). However, no satisfactory reference data regarding weight for height are available. For children over 9 years of age, skinfold thickness measurements are particularly desirable because of the lack of any suitable alternative for evaluation of obesity.

Screening Followup: Excess Weight for Height

In the case of infants with weight for length greater than the 95th percentile, the emphasis should be placed on avoiding further excessive weight gain. With a caloric intake of approximately 90 to 95 kcal/kg/day, one may anticipate that gain in fat-free tissue will continue at a normal rate whereas increase in body content of fat will not occur or will occur only at a slow rate. The major aim should be directed to correction of any patterns of overeating that may have been associated with the previous abnormal gain in weight.

A modification of this approach for preschool children may be effective also. As with infants, close medical supervision is essential.

Children of school age with weight for height greater than the 95th percentile should be referred to an obesity clinic or other treatment facility for further medical evaluation and treatment.

Head Circumference

The growth of head circumference up to two years of age is so closely related to growth of body length in malnutrition that head circumference measurements add no more information about a child's nutritional status than do body length measurements (Malina et al., 1975). After two years of age, head circumference grows so slowly that it is a poor indicator of actual malnutrition although in an older child it may be a good indicator of past malnutrition (i.e., during the first two years of life). Therefore, head circumference is not a useful screening measurement for actual malnutrition when body length or height measurements are known. For this reason head circumference is not recommended for screening children for malnutrition. Head circumference remains, as always, an important screening measurement for micro- and macrocephaly due to non-nutritional abnormalities.

Skinfold Thickness

As will be discussed in Part II, Chapter 1, obesity is a condition in which the amount of fat in the body is excessive in relation to total body weight. The relation of weight to height (or length), which is commonly employed as an index of fatness, is unfortunately unable to discriminate between an individual who is heavy because of large muscle mass and one who is heavy because he is obese. Skinfold thickness, on the other hand, appears to be rather well correlated with fat content of the body, at least in 8½- to 12-year-old children (Parízková and Roth, 1972). It seems likely that amounts of subcutaneous and intra-abdominal fat are more closely related in children than in adults and therefore skinfold thickness may be a better index of body fat in children than in adults.

Measurement of skinfold thickness requires attention to detail and considerable practice. Difficulty in making reproducible measurements of skinfolds in children (i.e., beyond infancy) is not appreciably greater than in making reproducible measurements of height in toddlers. Because of the difficulty, many programs will not find it feasible to measure skinfolds during the first two or three years of life and may wish to limit measurements in other age groups to children with weight for height greater than the 90th percentile value.

For measurement of skinfold thickness of preschool children, the Lange[1] caliper is preferred. This caliper meets the recommendations of the Committee on Nutritional Anthropometry (1956), and the area included within the jaws (30 mm²) is small enough to be practical for use with infants. Methods of measurement have been described by the

[1] Lange skinfold caliper, Cambridge Scientific Industries, Inc., Cambridge, Maryland 21613.

Figure 12 Identification of level of measurement of triceps skinfold. Note from Figure 13 that measurement at this level is made in midline posteriorly.

Figure 13 Measurement of triceps skinfold (see text).

Figure 14 Identification of site for measurement of subscapular skinfold.

Figure 15 Measurement of subscapular skinfold (see text).

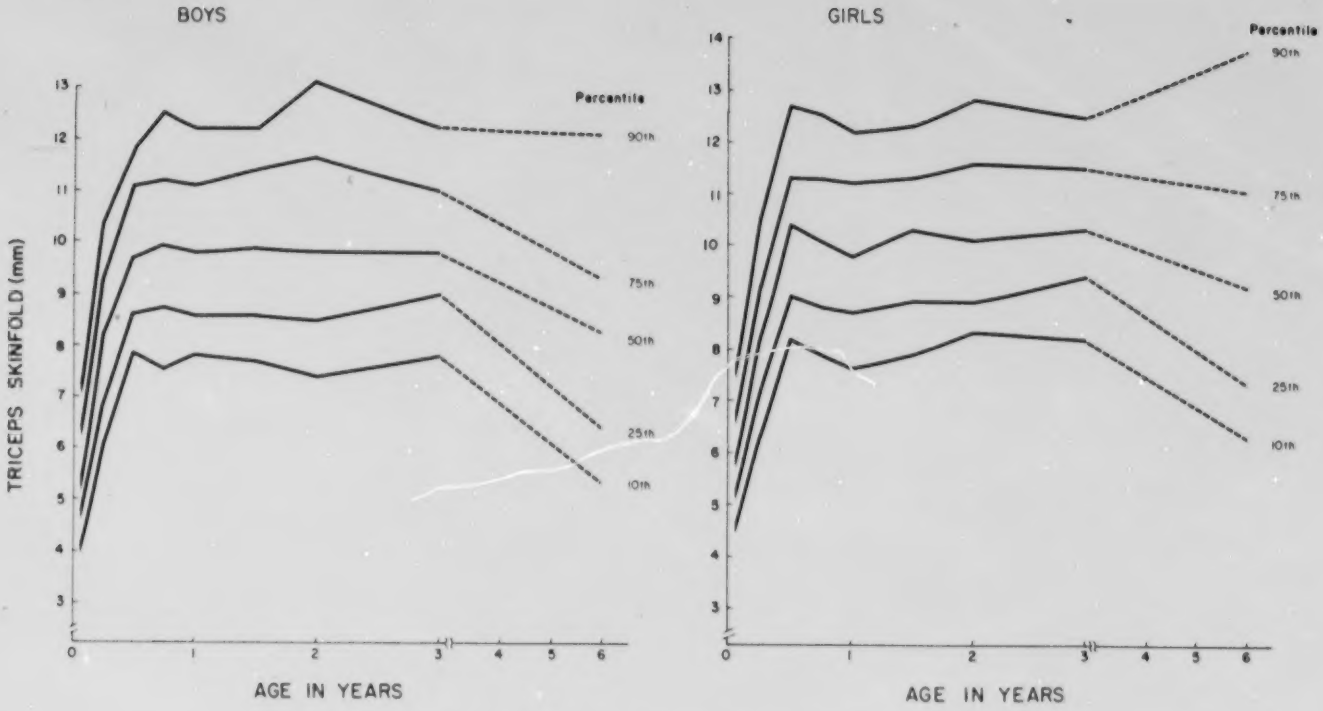

Figure 16 Triceps skinfold thickness of boys and girls less than 6 years of age (see text).

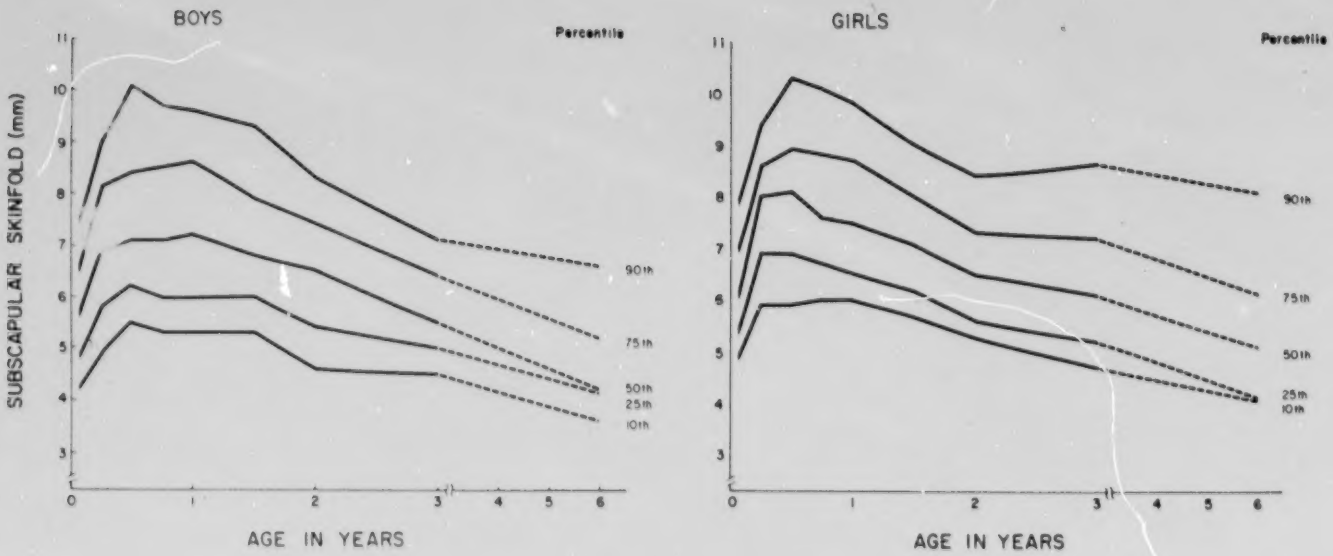

Figure 17 Subscapular skinfold thickness of boys and girls less than 6 years of age (see text).

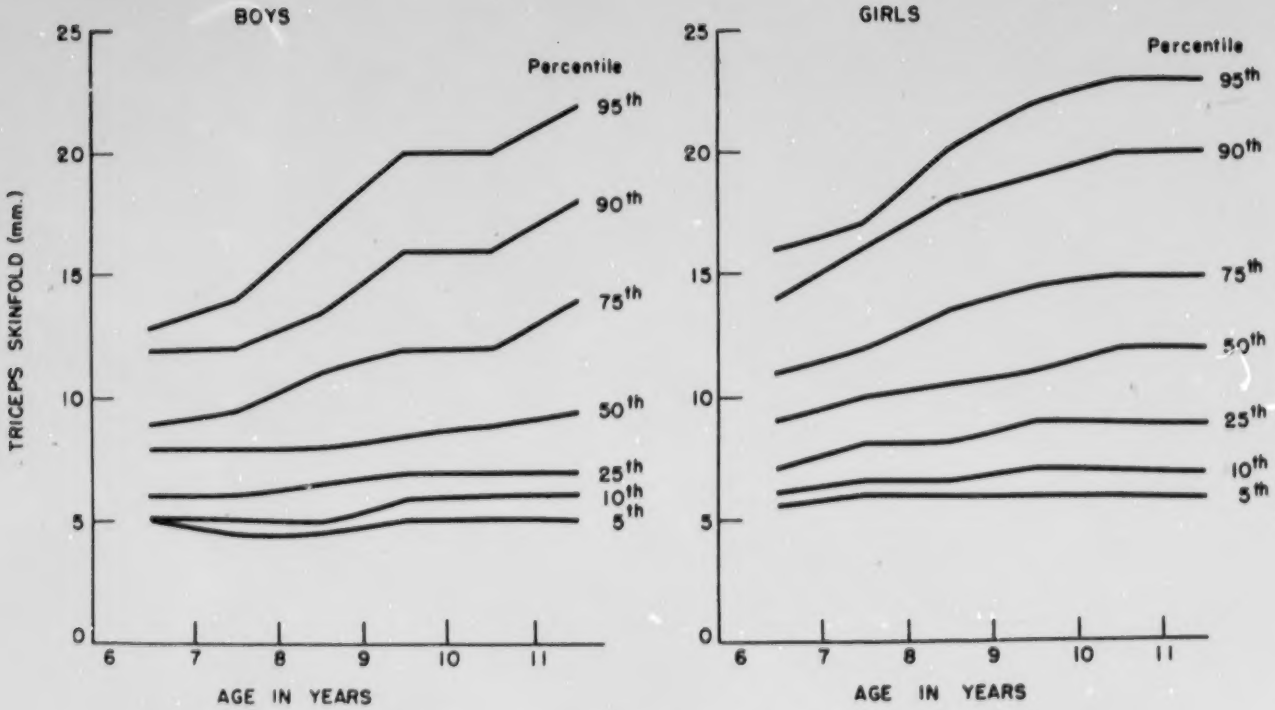

Figure 18 Triceps skinfold thickness of boys and girls 6 through 11 years of age (Johnston et al., 1972).

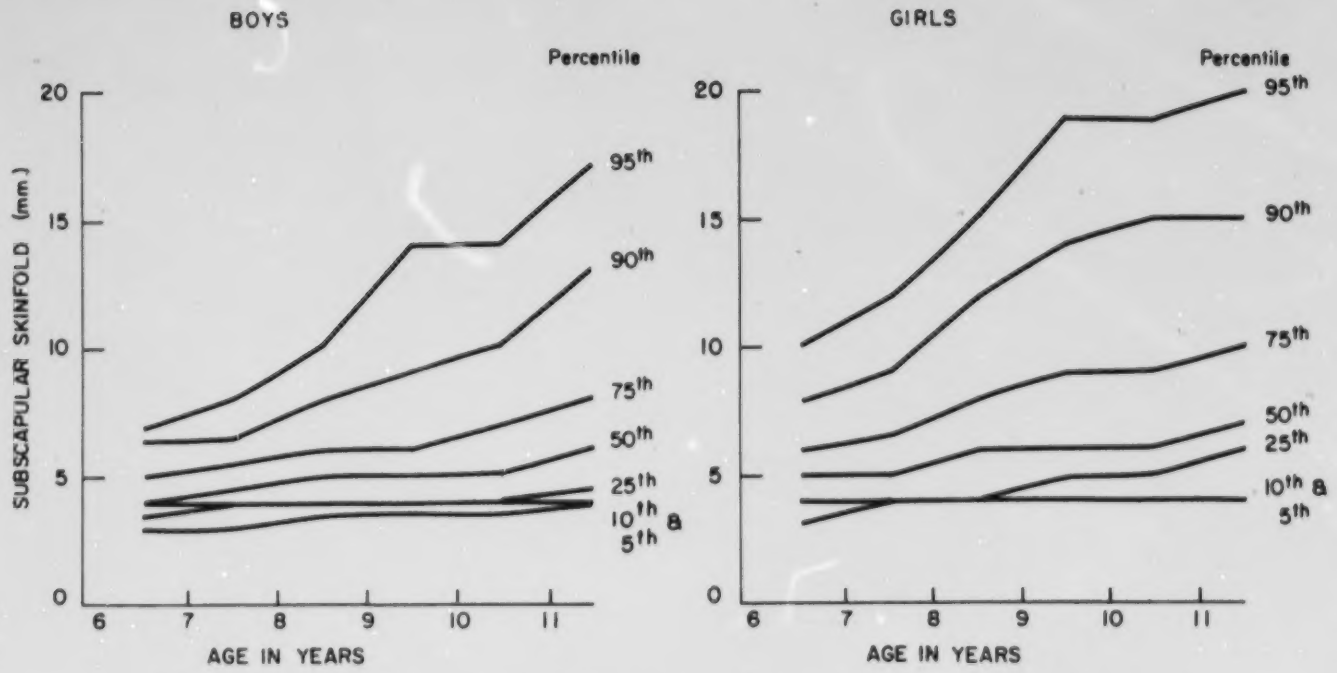

Figure 19 Subscapular skinfold thickness of boys and girls 6 through 11 years of age (Johnston et al., 1972).

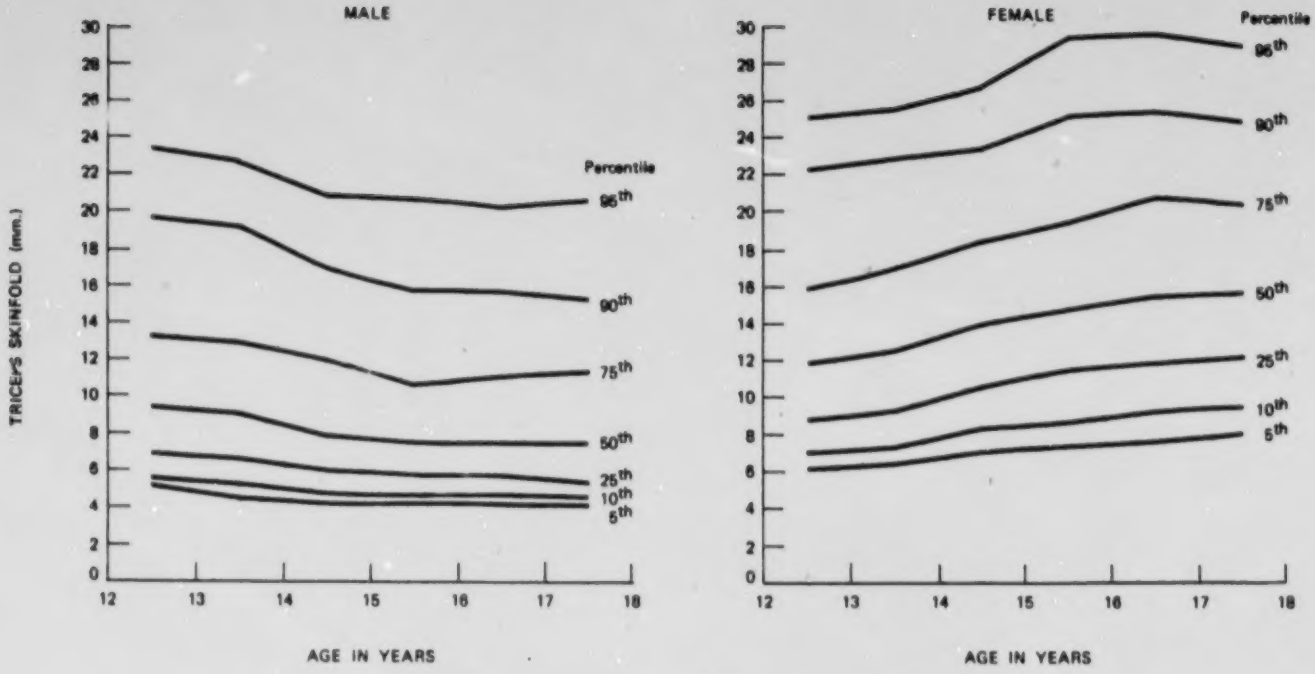

Figure 20 Triceps skinfold thickness of males and females 12 to 18 years of age (Johnston et al., 1974).

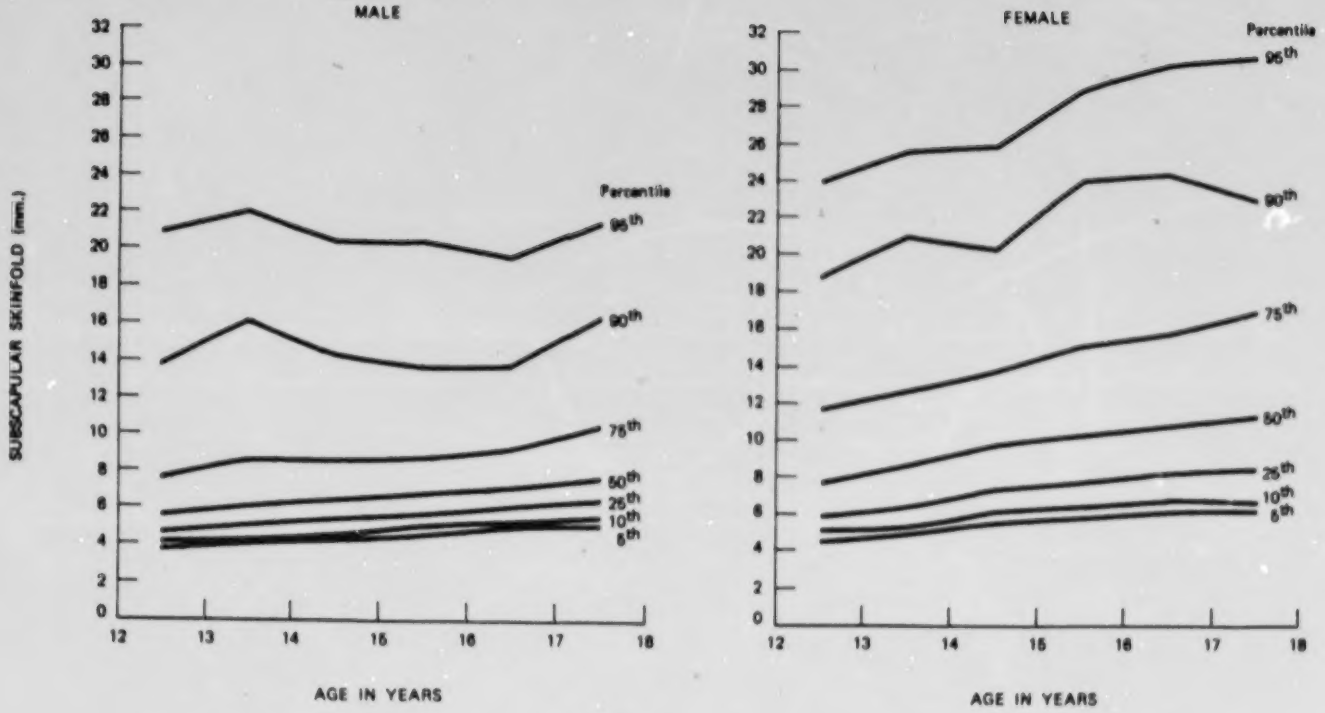

Figure 21 Subscapular skinfold thickness of males and females 12 to 18 years of age (Johnston et al., 1974).

Committee on Nutritional Anthropometry (1956) and by Jelliffe (1966). After age three years, the Lange caliper may still be used although for older children and adults many anthropometrists prefer the Harpenden caliper.*

Measurements at a variety of sites are useful but those most commonly utilized are triceps and subscapular. Skinfold measurements of infants are particularly difficult and require patience and continuing practice. The infant should be held in the lap of the attendant (preferably the mother) in a semi-upright position, his right side adjacent to but not touching her body and his head facing forward. The infant's left hand or forearm should be gently restrained with the elbow flexed to approximately 90° and the forearm pressed gently against the infant's abdomen. A child beyond infancy should be measured while standing with his arm in a relaxed position and the elbow extended.

For measurement of triceps skinfold thickness, marks are placed at the left acromion and olecranon (Fig. 12). The distance between these marks is measured and the midpoint marked. The skinfold is measured at the level of the marked midpoint in the posterior midline of the arm (Fig. 13). Subscapular skinfold thickness is measured just inferior and lateral to the inferior angle of the left scapula in the line of the natural skin cleavage (Fig. 14, 15).

At a site 1 cm from the measurement site, the examiner grasps the layer of skin and subcutaneous tissue with the first finger and thumb of one hand, pulling it away from the underlying tissue, and continues to hold it until the measurement is completed. Readings should be made to 0.1 mm approximately three seconds after application of the caliper.

Interpretation

Figures 16–21 present percentile values for triceps and subscapular skinfold thickness in relation to age. The data pertaining to the first three years of life (Fig. 16, 17) are those of Karlberg et al. (1968), concerning 120 male and 90 female Swedish children followed longitudinally. The 10th to the 90th percentile values are indicated. There are no American data of similar quality and number for the first three years of life and, unfortunately, there are no satisfactory data from any source concerning children 3 to 6 years of age. Percentile values for 3- to 6-year-old children included in Figures 16 and 17 represent interpolations between the values at 3 years of age reported by Karlberg et al. (1968) and the values of 6½ years of age from Figures 18 and 19.

Figures 18 and 19 present data from measurements of 7119 children 6 through 11 years of age from the U.S. Health Examination Survey (HES), Cycle II, 1962–65 (Johnston et al., 1972). These values are

*Harpenden skinfold caliper, H. E. Morse Co., 455 Douglas Avenue, Holland, Michigan 49423.

representative of 12.1 million males and 11.7 million females in this age group. Figures 20 and 21 present data from measurements of 6763 individuals 12 through 17 years of age from HES, Cycle III, 1966–70 (Johnston et al., 1974). The values are representative of 11.5 million males and 11.2 million females in this age group. The 5th and 95th percentile values from the Health Examination Survey data are included. (The 5th and 95th percentiles are not available in the report by Karlberg et al., 1968).

Screening Followup

When the triceps or subscapular skinfold thickness is greater than the 90th percentile value for age, close medical supervision and counseling are desirable. When skinfold thickness exceeds the 95th percentile value, referral to an obesity clinic or other treatment facility will usually be desirable. Because satisfactory 95th percentile values are not available for the first 6 years of life, it will be necessary to utilize some arbitrary value as an indication for referral for treatment. A value 1 mm greater than the 90th percentile is a reasonable value on which to base referral. At most ages, this value will be an approximation of the 95th percentile.

Laboratory Studies

Laboratory studies are of great assistance in screening children for nutritional disorders. Seldom will it be feasible to perform the full range of determinations listed in Table I. In most instances it will be desirable to select a few laboratory studies to be carried out routinely and to do other studies only with children suspected on clinical grounds of specific disorders.

As a minimum, it is suggested that hemoglobin concentration or hematocrit be determined. If feasible, serum concentration of cholesterol should also be determined. The desirability of performing other laboratory studies on a routine basis will depend in part on the level of laboratory competence available locally or the ease with which arrangements can be made for laboratory studies to be performed at a more distant site.

Certain laboratory determinations are likely to be of greatest value in one community while other determinations may be more valuable in another community. Within the same community, priorities may vary from year to year or even within a period of several months. In general, specific analyses will be selected on the basis of clues provided by knowledge of the community, information about food intake or by physical findings.

For example, in a cloudy city where milk that has not been fortified with vitamin D is readily available for purchase, screening of early pre-

TABLE I

BIOCHEMICAL METHODS AND REMARKS REGARDING INTERPRETATION

Substance	Method	Quantity Required	Comment
Hemoglobin (blood)	Cyanmethemoglobin (O'Brien et al., 1968a)	20 μl	Concentration of hemoglobin less than 11.0 g/100 ml for children below 10 years of age and less than 12.0 g/100 ml for older children (less than 13.0 g/100 ml for males over 14 years of age) indicates anemia (See Part II, Chap. 4).
Hematocrit (blood)	Capillary tube (O'Brien et al., 1968b)	40 μl	Hematocrit less than 34 for children below 10 years of age and less than 37 for older children (less than 41 for males over 14 years of age) indicates anemia (see Part II, Chap. 4).
Iron and iron-binding capacity (serum)	Manually by method of Fischer and Price (1964) or automated (Garry and Owen, 1968)	200 μl 100 μl	Concentration of iron, iron-binding capacity and percent saturation of transferrin may require different interpretation in infants than in older individuals (See Part II, Chap. 4).
Free erythrocyte porphyrins (blood)	Method of Piomelli et al. (1976) with filter paper disc	100 μl	Free erythrocyte porphyrin/hemoglobin ratio greater than 5.5 μg/g indicates iron deficiency.
Total protein (serum)	Microbiuret manually (O'Brien et al., 1968c) or automated (Failing et al., 1970)	50 μl	With manual method, a serum blank is desirable.
Albumin (serum)	Electrophoresis on cellulose acetate (Fomon et al., 1970)	10 μl	Concentration of albumin less than 2.9 g/100 ml suggests poor protein nutritional status.
Ascorbic acid (plasma)	2,6 Dichloroindophenol reaction manually (O'Brien et al., 1968d) or automated (Garry et al., 1974)	20 μl 50 μl	Concentration less than 0.3 mg/100 ml suggests that recent dietary intake has been low.

Vitamin A (plasma or serum)	Fluorometry (Garry et al., 1970; or Thompson et al., 1971)	200 μl	Concentration less than 10 μg/100 ml suggests deficiency and concentration less than 20 μg/100 ml indicates low stores.
Alkaline phosphatase (serum)	Liberation of p-nitrophenol manually (O'Brien et al., 1968e) or automated (Morgenstern et al., 1965)	100 μl	Activity greater than 25 Bodansky units/100 ml is suggestive of rickets.
Inorganic phosphorus (serum or plasma)	Modification of method of Fiske and Subba Row (1925) manually (O'Brien et al., 1968f) or automated	50 μl	Concentration less than 4.0 mg/100 ml is abnormal and suggestive of rickets. However, normal concentration does not rule out the presence of rickets.
Urea nitrogen (serum)	Urease manually (O'Brien et al., 1968g) or diacetyl monoxime manually or automated (Marsh et al., 1965)	100 μl 50 μl	Concentration less than 8 mg/100 ml suggests low recent dietary intake of protein. However, concentrations as low as 3.5 mg/100 ml are sometimes found in breastfed infants.
Cholesterol (serum)	Manually by method of Carr and Drekter (1956) or automated (Levine and Zak, 1964)	100 μl	Concentration of cholesterol more than 230 mg/100 ml indicates hypercholesterolemia (See Part II, Chap. 2).
Lipoproteins (serum)	Agarose electrophoresis (Laboratory Methods Committee, 1974)	100 μl	For interpretation, see Fredrickson and Levy (1972).
Creatinine (urine)	Alkaline picrate manually (O'Brien et al., 1968h) or automated	100 μl	Serves as reference for other urine determinations.
Riboflavin (urine)	Fluorometry (Horwitz, 1970a)	2 ml	Excretion less than 250 μg/g of creatinine suggests low recent dietary intake.
Thiamin (urine)	Thiochrome fluorometry (Horwitz, 1970b)	10 ml	Excretion of less than 125 μg/g of creatinine suggests that dietary intake has been low for weeks or months.
Iodine (urine)	Automated ceric ionarsenious acid system (Garry et al., 1973)	5 ml	Excretion of less than 50 μg/gm of creatinine suggests low recent dietary intake.

school children for rickets might receive highest priority. For at least several months, preferably including the late winter, alkaline phosphatase activity might be determined in sera of all children less than 3 years of age; roentgenograms of the wrist might be made of all children with elevated alkaline phosphatase activity or with the slightest clinical suggestion of rickets. When several hundred children have been screened in this manner, it should be possible to decide whether or not further screening and preventive measures are desirable. If rickets does not appear to be a problem of major importance in the community, routine screening should probably be discontinued.

Procedures

In most instances, it will be desirable to obtain a small sample of venous blood from the antecubital or external jugular vein (the femoral vein is not recommended). However, several of the determinations listed in Table I can be performed on an amount of capillary blood readily obtained from fingertip or heel. Blood should be placed immediately into plain tubes (for serum) or into dry, heparinized tubes (for whole blood or plasma).

Hematocrit and concentration of hemoglobin should be determined with blood obtained directly from the fingertip or heel or from the heparinized tube. Heparinized blood should be centrifuged immediately and non-heparinized blood should be centrifuged as soon as it has clotted. If concentration of ascorbic acid is to be determined, it should be performed immediately or the plasma should be mixed with metaphosphoric acid and frozen (without separating the supernatant from the protein precipitate) until the determination can be performed. Other determinations should be performed within eight hours or the serum or plasma should be frozen and maintained in the frozen state until the analyses can be performed.

Urine should be acidified with hydrochloric acid to a final pH less than 3.0. If analyses cannot be carried out within eight hours, the samples should be stored in the frozen state.

Iron Nutritional Status

In the United States and in most other countries, low concentrations of hemoglobin of infants and preschool children are most commonly an expression of iron deficiency. For reasons to be discussed in Part II, Chapter 4, anemia is defined as a condition in which hemoglobin concentration is less than 11.0 gm/100 ml in children below 10 years of age, or less than 12 gm/100 ml in older children or adolescents with the exception of males over 14 years of age. For this latter group a hemoglobin concentration less than 13.0 gm/100 ml is assumed to in-

dicate anemia. Hematocrit values of 34, 37 and 41 correspond roughly with hemoglobin concentrations of 11, 12 and 13 gm/100 ml.

As will be discussed in Chapter 4 (p. 96), at least three practical approaches are available for determining whether anemia is associated with iron deficiency: (1) the response of a low hemoglobin concentration to administration of iron; (2) saturation of transferrin less than 16 or 17%; (3) ratio of free erythrocyte porphyrins (FEP) to hemoglobin greater than 5.5 μg per gram when lead concentration of the blood is less than 40 μg/100 ml. Increase in concentration of FEP occurs because heme synthesis cannot be completed in the absence of an adequate supply of iron. A relative excess of unutilized ("free") protoporphyrin therefore accumulates in the erythrocytes. The presence of an excess body burden of lead interferes with incorporation of iron into heme and therefore also causes an increased FEP to hemoglobin ratio. Concentrations of lead in blood less than 40 μg/100 ml are rarely responsible for increased concentration of FEP (Piomelli et al., 1973).

SCREENING FOLLOWUP:

When hemoglobin concentration or hematocrit is less than the values indicated in the previous section, the history of iron intake should be reviewed in detail and the laboratory value should be repeated for confirmation. With black children, a test for sickle cell anemia should be carried out. When feasible, serum concentration of iron and percent saturation of transferrin and/or the FEP-hemoglobin ratio should be determined. When it is impractical to carry out additional hematologic studies (and in the presence of a negative sickle cell test in black children), a trial of administration of iron—30 mg daily of ferrous sulfate or other well-absorbed form of medicinal iron—should be conducted. Repeat determinations of hemoglobin (or hematocrit) should be made at intervals of 4 weeks. If evidence of improved hematologic status is not observed, complete hematologic evaluation is desirable.

It is important to note that the definition of anemia is based on values that are largely arbitrary. Infants and children less than 2 years of age will not uncommonly be found to have hemoglobin concentrations between 10.5 and 11.0 gm/100 ml. For these infants and children, the diet history should be reviewed, dietary advice provided, and another determination of hemoglobin concentration scheduled in about 6 months. Similarly, children 10 to 12 years of age with hemoglobin concentrations between 11.5 and 12.0 g/100 ml and males 14 to 16 years of age with hemoglobin concentrations between 12.0 and 13.0 g/100 ml do not necessarily call for vigorous followup. Such children should, however, be followed more closely than those with greater hemoglobin concentrations.

In some clinics hemoglobin concentration less than 10.5 or even less than 10.0 g/100 ml will be found in a high percentage of younger chil-

dren. These clinics should, of course, direct their efforts to this more seriously affected group while instituting preventive measures in the community. In this instance, as with abnormalities of other parameters, followup activities should be directed toward the individuals at greatest risk.

Serum Cholesterol Screening

As will be discussed in Part II, Chapter 2, elevated serum concentration of cholesterol in adults is associated wth increased risk of coronary heart disease and stroke. Considerable evidence suggests that atherosclerosis, the abnormality that leads to coronary heart disease and stroke, begins in childhood. It is likely that increased serum concentrations of cholesterol during childhood will contribute to more rapid than usual progression of the atherosclerotic process. Thus, vigorous screening efforts seem desirable in order to identify the children at greatest risk. The relation between serum concentration of cholesterol and risk with respect to coronary heart disease and stroke is discussed more fully in Part II, Chapter 2.

SCREENING FOLLOWUP:

Children with serum cholesterol concentrations between 200 and 230 mg/100 ml, verified by a second determination, deserve closer medical observation than do children with concentration less than 200 mg/100 ml. When feasible, families of these children should be screened; dietary supervision and further determinations of serum concentrations of cholesterol at approximately yearly intervals are desirable.

Serum concentrations of cholesterol greater than 230 mg/100 ml (verified by a second determination) should be considered distinctly abnormal and a strong indication for additional study and treatment. Studies should include determination of serum concentrations of triglycerides and serum lipoprotein profile (Fredrickson and Levy, 1972) (both determined with blood obtained during fasting) as well as screening of other family members for serum cholesterol concentration. Dietary management is discussed in Part II, Chapter 2.

Screening Followup of Other Biochemical Findings

Values outside the ranges arbitrarily defined as normal (Table I) may be accepted as presumptive evidence of abnormality. In each case, the total information about the child should be reviewed in the light of the abnormal biochemical value and all relevant tests repeated for confirmation. Appropriate referral for treatment or advice can then be made.

As an example, one may consider the finding of an abnormally low serum concentration of albumin. If the result is confirmed with a sec-

ond specimen of blood, further evaluation would be desirable. The history would be reviewed, particularly with respect to intake of protein during the previous weeks or months and in relation to signs or symptoms suggesting renal, hepatic or gastrointestinal disease, including cystic fibrosis of the pancreas. Serum concentration of urea nitrogen would be determined as an indication of recent dietary intake of protein and/or renal disease, and urine would be examined for albumin. With this evidence at hand, one would be in a position to decide on the desirability of additional studies or dietary treatment.

Roentgenograms

Whenever clinical or biochemical evidence suggests rickets or scurvy, it is desirable to confirm the finding by roentgenograms of the wrist. It is not recommended that roentgenograms of the wrist be made routinely in screening unless (1) a roentgenologist experienced in interpreting early evidences of rickets and scurvy is available, (2) biochemical studies of plasma routinely include determinations of ascorbic acid, inorganic phosphorus and alkaline phosphatase. Routine roentgenograms are of value only during the first two years of life, when rickets is most prevalent.

Summary

The frontispiece summarizes the screening process. If a child presents no suspicion of nutritional disorder on the basis of the various screening approaches—i.e., the food and diet questions, physical examination, anthropometric examination and laboratory studies—it will be concluded that there is little likelihood of detecting a nutritional abnormality. The child (or his family) will then be given advice regarding prevention of nutritional disorders and the screening process will be repeated in one or two years.

If a suspicion of nutritional disorder arises from any of the screening approaches, some additional evaluation will be desirable. If answers to the screening questions suggest that habits of eating are unusual or that the diet itself is unusual or monotonous, an interview with a competent nutritionist should be arranged. If this interview provides additional evidence of inadequate diet, further study (for example, specific laboratory tests) and/or dietary counseling will be indicated. If no evidence of dietary inadequacy is found in this interview and if the child also "passes" the other phases of screening (physical examination, anthropometric evaluation and laboratory studies), the child will be considered to have passed the screen and further evaluation will not be necessary.

When suggestive evidence of nutritional deficiency is detected by physical examination, appropriate laboratory studies should be undertaken to determine whether a nutritional disorder actually exists (see Table I).

In some instances (for example, clinical signs suggesting deficiency of B vitamins) an interview with a nutritionist will be desirable in addition to, or in lieu of, laboratory studies. Whenever laboratory findings confirm clinical signs of nutritional deficiency, an interview with a nutritionist is essential. The nutritionist and physician should then review the findings and decide whether dietary counseling (Bureau of Community Health Services, 1976) and followup are adequate for management or whether additional studies and perhaps hospitalization are desirable to rule out the presence of specific diseases (for example, renal osteodystrophy).

As already discussed, excessive weight for height or skinfold thickness for age are indications for intervention. However, intervention measures have little likelihood of success unless the child is highly motivated. In the case of younger children, there is particular need for the family to cooperate with the physician and to support any program of weight control that is attempted. The nutritionist can often aid the physician in eliciting cooperation of the family.

When height for age or weight for height is less than the 5th percentile value, evaluation by a physician will be desirable. Special attention should be directed to the history and physical examination in order to detect any evidence of cardiovascular, renal, hepatic or other abnormality. When physical examination does not provide clues concerning an abnormality, an interview with a nutritionist is desirable and simple laboratory studies should be carried out. At times, hospitalization may be necessary. Deficient dietary intake should be corrected if possible. If dietary intake is deficient or if no cause is found for the short stature or low weight for height, the child should be reexamined at approximately six-month intervals. Children who remain below the 5th percentile of height for age (or weight for height) but who seem otherwise normal and whose growth progresses in parallel with that of normal children need not be subjected to extensive diagnostic studies.

When hemoglobin is less than 11.0 g/100 ml for children less than 10 years of age or less than 12.0 g/100 ml for older children and adolescents (less than 13.0 g/100 ml for males 14 years of age or older)* additional hematologic evaluation and an interview with a nutritionist to provide an estimate of usual intake of iron are desirable. The advisability of dietary modification and/or specific treatment can then be determined. When it is not feasible to carry out hematologic evaluation, a trial with iron therapy (e.g., 30 mg of elemental iron in the form of ferrous sulfate or other well-absorbed iron) may be carried out with followup at monthly intervals. However, as already mentioned, black children should not be so treated unless the sickle cell test is negative. When low concentration of hemoglobin persists in spite of iron therapy, referral for additional hematologic evaluation is mandatory.

*Hematocrit values of 34, 37 and 41 correspond approximately to hemoglobin concentrations of 11.0, 12.0 and 13.0 g/100 ml, respectively.

When serum concentration of cholesterol is greater than 200 mg/100 ml, the value should be confirmed by a second determination. It is preferable to obtain the sample of blood for the second determination with the child in the fasting state and to include determination of serum concentration of triglycerides as well as cholesterol. If cholesterol concentration is between 200 and 230 mg/100 ml, yearly followup is desirable. In instances in which serum concentrations of cholesterol are consistently greater than 230 mg/100 ml, lipoprotein electrophoresis should be carried out, weight control measures should be instituted if the child is overweight, and dietary changes should be instituted, as discussed in Part II (Chapter 2) of this publication.

When other laboratory values are outside of the normal range (Table I), the abnormal value should be repeated for confirmation and then additional studies or treatment should be instituted. The type of studies and/or treatment will depend on the specific biochemical abnormality.

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APPENDIX TO PART I
INTERVIEW BY NUTRITIONIST

Name_____
Date & Time of Interview_____
Length of Interview_____
Date of Recall_____
Day of the week of Recall
1–M 2–T 3–W 4–Th 5–F 6–Sat 7–Sun

"I would like you to tell me about everything your child ate and drank from the time he got up in the morning until the time he went to bed at night and what he ate during the night. Be sure to mention everything he ate or drank at home, at school, and away from home. Include snacks and drinks of all kinds and everything else he put in his mouth and swallowed. I also need to know where he ate the food, but now let us begin."

What time did he get up yesterday? _____
Was it the usual time? _____
What was the first time he ate or had anything to drink yesterday morning?
(list on the form that follows)
Where did he eat? (list on the form that follows)
Now tell me what he had to eat and how much?
(Occasionally the interviewer will need to ask:)
When did he eat again? or, is there anything else?
Did he have anything to eat or drink during the night?
Was intake unusual in any way? Yes_____ No_____
(If answer is yes) Why? _____
In what way? _____
What time did he go to bed last night? _____
Does he take vitamin and/or mineral supplements?
Yes _____ No _____
(If answer is yes) How many per day? _____
Per week? _____
What kind? (Insert brand name if known)
Multivitamins _____
Ascorbic Acid _____
Vitamins A and D _____
Iron _____
Other _____
Does he eat differently on weekends? Yes _____ No _____ (Describe the differences)
_____
Would you describe his appetite as Good? _____ Fair? _____ Poor? _____
What are his favorite foods? _____
What foods does he dislike? _____

What are his favorite snacks? _____

_____

Who prepares your child's food? (e.g., mother, grandmother, baby sitter, brother, sister, etc.) _____

Who feeds him? _____

Does he eat alone? Yes_____ No_____

Does he participate in a school lunch, school breakfast, WIC or other feeding program? Yes_____ No_____

Is he on a special diet now? Yes_____ No_____

If Yes, why is he on a diet? (check)

_____for weight reduction (own prescription)

_____for weight reduction (doctor's prescription)

_____for gaining weight

_____for allergy, specify _____

_____for other reason, specify _____

If No, has he been on a special diet within the past year? Yes_____ No_____

If Yes, for what reason? _____

Does he eat anything which is not usually considered food? (e.g., dirt)

Yes_____ No_____

If yes, what? _____

how often? _____

What? _____

How often? _____

Has a doctor or nurse ever told you that he was anemic (had low blood)?

Yes_____ No_____

Does he have frequent problems with constipation or diarrhea?

Yes_____ No_____

Does he complain of sore gums or aching teeth? Yes_____ No_____

Does he have trouble chewing or swallowing? Yes_____ No_____

Is he allergic to any foods? Yes_____ No_____

Does he take any medication regularly? _____

Can he feed himself? Yes_____ No_____

(with his fingers? _____ with a spoon? _____)

Can he use a cup or glass by himself? Yes_____ No_____

Does he drink from a bottle with a nipple? Yes_____ No_____

Use of food frequency record

Indicate whether or not the child ate the following foods by checking the columns "does not eat" or "does eat" for each item. For each food checked "does eat" write the approximate number of times eaten in a week. If any particular food is eaten less than once a week, do not write anything in the column "times eaten per week".

In some cases more than one food has been listed on a line. If the child does not eat all of these foods, underline the specific food(s). A space has been provided at the end to write in foods not listed which are regularly eaten.

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FOOD FREQUENCY RECORD*

	Food	Does Not Eat	Does Eat	Times Eaten Per Week
I.	Chicken			
	Beef, hamburger, veal			
	Liver, kidney, tongue, etc.			
	Lamb			
	Cold cuts, hot dogs			
	Pork, ham, sausage			
	Bacon			
	Fish			
	Kidney beans, pinto beans, lentils (all legumes)			
	Soybeans			
	Eggs			
	Nuts or seeds			
	Peanut butter			
	Tofu			
II.	Milk (fluid, dry, evaporated)			
	Cottage cheese			
	Cheese (all kinds other than cream)			
	Condensed milk			
	Ice cream			
	Yogurt			
	Pudding and custard			
	Milkshake			
	Sherbert			
	Ice milk			
III.	Whole grain bread			
	White bread			
	Rolls, biscuits, muffins			
	Bagels			
	Crackers, pretzels			
	Pancakes, waffles			
	Cereals, incl. grits			
	White rice			

* Modified slightly from California Department of Health (1975).

	Food	Does Not Eat	Does Eat	Times Eaten Per Week
	Brown rice			
	Noodles, macaroni, spaghetti			
	Tortillas (flour)			
	Tortillas (corn)			
IV.	Tomato, tomato sauce, or tomato juice			
	Orange or orange juice			
	Tangerine			
	Grapefruit or grapefruit juice			
	Papaya, mango			
	Lemonade			
	White potato			
	Turnip			
	Peppers (green, red, chili)			
	Strawberries, cantaloupe			
V.	Lettuce			
	Asparagus			
	Swiss chard			
	Cabbage			
	Broccoli			
	Brussels sprouts			
	Scallions			
	Spinach			
	Greens (beet, collard, kale, turnip, mustard)			
VI.	Carrots			
	Artichoke			
	Corn			
	Sweet potato or yam			
	Zucchini			
	Summer squash			
	Winter squash			
	Green peas			
	Green and yellow beans			
	Hominy			
	Beets			
	Cucumbers or celery			

	Food	Does Not Eat	Does Eat	Times Eaten Per Week
	Peach			
	Apricot			
	Apple			
	Banana			
	Pineapple			
	Cherries			
VII.	Cakes, pies, cookies			
	Sweet roll, doughnuts			
	Candy			
	Sugar, honey, jam, jelly			
	Carbonated beverages (sodas)			
	Coffee or tea			
	Cocoa			
	Fruit drink			
VIII.	Other foods not listed which the child regularly eats			

Suggestions for Interviewers

Information will usually be obtained from the person responsible for feeding the child. Older children may be able to give more reliable information regarding their own intakes than will the responsible adult. The interviewer should judge this in each individual case.

How questions are asked is important. Avoid questions that suggest the correct answers—e.g., Did you have a dark-green or deep-yellow vegetable today? Avoid expressing approval or disapproval of the foods reported. If you feel there are omissions, ask additional questions: What did he drink with his lunch? What did he have on his toast?

Check carefully for the following information:

Additions to foods already recorded, such as:

1. Fats: Butter, margarine, honey-butter, peanut butter, mayonnaise, lard, meat drippings, cheese spreads, and others.
   Used on toast, bread, rolls, buns, cookies, crackers, sandwiches.
   Used on vegetables.
   Used on potatoes, rice, noodles, etc.
   Used on other foods.
2. Sugars: Jam, jelly, honey, syrup, sweetening, etc.
   Used on breads, sandwiches, vegetables, fruit, cereal, coffee, tea, other foods.
3. Other spreads: Catsup, mustard, etc.
4. Milk: Cream, half and half, skim milk, etc.
   Used on cereal, coffee, tea, desserts, other foods.
5. Gravies: Used on bread, biscuits, meat, potatoes, rice, noodles, other foods.
6. Salad dressings: Used on vegetables, salads, sandwiches, other foods.
7. Chocolate or other flavoring to milk, e.g., Quik, Bosco.

Food preparation

1. Preparation of eggs, e.g., fried, scrambled, boiled, poached.
2. Preparation of meat, poultry, fish, e.g., fried, boiled, stewed, roasted, baked, broiled.
3. Preparation of mixed dishes—major ingredients used, e.g., tuna fish and noodles, macaroni and cheese.
4. Special preparation of food—strained, chopped, etc.

Special additional detail about food items

1. Kinds of milk (whole, partially skim, skim, powdered, chocolate, etc.)
2. Kinds of carbonated beverages (regular, low-calorie).
3. Kinds of fruits (canned, frozen, fresh, dried, cooked with sugar added).
4. Kinds of fruit juices, fruit drinks, or juice substitutes.

By carrying a few standardized props* it will be possible to obtain more accurate recording of amounts: a teaspoon and tablespoon; several sizes of glasses and bowls (including a 4-oz and an 8-oz measure); something to indicate thickness of meat—a ruler or a standard form such as a model of a slice of bread.

* An assortment of props is available from: Nasco, Fort Atkinson, Wisconsin 53538, or 1524 Princeton Ave., Modesto, California 94352.

PART II
PREVENTION OF COMMON DISORDERS

Although screening and followup, as outlined in Part I, are an essential component of health care delivery, they cannot alone be considered adequate. Major efforts must be directed at prevention of disease. In some instances—for example, obesity—efforts at treatment have been remarkably unsuccessful. In others—for example, dental caries—the cost of treatment in discomfort, time, and money is enormous.

In high quality health care delivery, the approaches to screening, followup and prevention are carried out concurrently, and there is often no clear demarcation between the effort at screening or followup and the effort at prevention. In screening, a child with an inadequate diet may be identified and the followup will include counseling. In this case, followup and prevention will be the same. Nevertheless, some nutritional disorders are so prevalent and so complex that it seemed desirable to devote specific sections to them. Part II therefore presents discussions of obesity, atherosclerosis, dental caries and iron-deficiency anemia.

CHAPTER 1
PREVENTION OF OBESITY

SAMUEL J. FOMON and EKHARD E. ZIEGLER

Definition

Obesity may be defined in non-specific terms as an excessive ratio of fat to fat-free body mass. Although there is no convenient method of obtaining reliable estimates of total body content of fat, an estimate suitable for clinical purposes may be obtained from measurements of skinfolds; and at least strong clues may be obtained from simple measurements of height and weight. However, even with a satisfactory estimate of body content of fat, one is left with the problem of defining what is an excessive ratio of fat to fat-free body weight.

In the adult, as the ratio of body weight to body height increases above the average for the population, the prevalence of diabetes mellitus, hypertension and atherosclerosis increases progressively (Mann, 1974). However,there is no sharp cutoff point in the relation of weight to height below which these abnormalities are uncommon and above which they are common. Thus, the establishment of a medically-based definition of obesity is difficult in the adult.[10] In the child, for whom the medical significance of obesity lies primarily in the likelihood that obesity will persist into adulthood, a medically satisfying definition of obesity is even more difficult to develop and, in fact, at present appears unattainable.

In the absence of a medically-based definition, an operational definition is used that will presumably identify at least those children who are most obese. As already mentioned (page 45) triceps and/or subscapular skinfold thickness greater than the 95th percentile of the reference data or weight for height greater than the 95th percentile value is considered evidence of obesity.

The problem of defining obesity in a medically meaningful way is further complicated by the possibility that normal individuals may dif-

[10] Tables of weight for height used by the life insurance industry have been prepared on the basis that "ideal weight" is defined as weight for height associated with the lowest mortality. Deviations from ideal weight indicate increased mortality risk and, therefore, insurance tables are, in a sense, medically meaningful.

fer in their ratios of fat to fat-free body weight. Perhaps some specified moderate degree of obesity may allow one individual to function optimally, while the same degree of obesity may constitute a health hazard for another.

Early Prediction of Later Obesity

Obesity, whether of children or adults, is discouragingly refractory to treatment. This is particularly true in early-onset obesity. It appears, therefore, that efforts aimed at prevention should receive primary attention. Early identification of the subject at risk, if feasible, would be invaluable in that it could reduce the target population to a more manageable size. It is therefore appropriate to ask whether the obesity-prone subject can be identified early in life.

Parental Obesity, Body Weight and/or Weight Gain During Infancy or Childhood

Obesity tends to cluster in families. With one obese parent, a child has a risk of about 40% of becoming an obese adult; with two obese parents, the risk increases to 70% (Bruch, 1973a). In predicting that a child will become obese, knowledge of parental obesity is almost certainly more useful than is knowledge of weight gain during infancy or of weight for height during childhood. How much of this familial trend is due to genetic factors and how much to environmental and social factors remains undetermined. These factors may render an individual more refractory to preventive and therapeutic efforts, but that does not mean that such efforts should be withheld; on the contrary, such individuals might benefit more than others from efforts at intervention.

As summarized elsewhere (Fomon, 1974a), a number of investigators have reported an association between weight gain or obesity in infancy and obesity in childhood. Unfortunately, several of the studies demonstrate deficiencies in experimental design or problems in interpretation. Mellbin and Vuille (1973), on the basis of one of the few studies with a satisfactory experimental design, concluded that "only 10–20% of the variation in weight for height (at age 7 years) can be explained by factors whose effect was detectable in infancy."

On the other hand, obesity in childhood has been shown to be associated strongly with obesity in adulthood (Mossberg, 1948; Mullins, 1958; Abraham and Nordsieck, 1960; Lloyd et al., 1961; Abraham et al., 1971; Miller et al., 1972). There can be little question that the obese child has a high likelihood of becoming an obese adult. Thus, parental obesity and obesity during childhood (beyond infancy) appear to be the major predictors of obesity in the adult.

Hyperplasia of Adipose Tissue

Excessive body fat is stored in adipose tissue. Obesity is therefore associated with increase in number and/or size of lipid-storing cells (adipocytes). In normal subjects there is a developmental increase of adipose cell number during infancy and childhood (Hirsch and Knittle, 1970); cell number characteristic of the adult is believed to be reached around 15 years of age (Brook, 1971). However, methods used for determining the total number of adipose cells in infants and children are likely to be highly inaccurate (Widdowson and Shaw, 1973; Garrow, 1974a; Fomon, 1974b) and such studies must be interpreted with great care.

Nevertheless, it seems possible on the basis of several studies (Hirsch and Knittle, 1970; Brook et al., 1972; Salans et al., 1973) that the earlier the onset of obesity the greater the degree of adipose tissue hyperplasia (increase in number of adipose cells). Much less certain is the meaning of this finding and whether hyperplasia, once established, is really irreversible. There appears to be little reason to conclude, as have several authors (Knittle, 1972; Brook et al., 1972), that overfeeding during early infancy or childhood leads to hyperplasia of adipose tissue. It seems unlikely that measurements of adipocyte size and number during infancy and childhood will aid greatly in prediction of later obesity.

Energy Intake and Expenditure

Because excessive gain in weight represents an imbalance between energy intake and energy needs, prevention of obesity cannot be concerned only with energy intake. Energy needs are determined by basal expenditures and by energy requirements for activity and growth. After infancy, energy requirements for growth represent a small percentage of total energy requirements. Basal expenditures vary from child to child but are more or less fixed in a particular child. The balance therefore depends primarily on the relation between energy intake and expenditures of energy in activity.

Imbalance between energy intake and expenditure leading to excessive gain in weight may result from abnormally high intake, unusually low expenditure, or from a combination of the two. Relatively small excesses of energy, accumulating day after day, year after year, are the common antecedent of obesity. For an adult to gain 5 kg of weight over a period of one year, a total excess of 30,000 kcal are required, or 82 kcal per day (Garrow, 1974b). A more common pattern, especially for children, is an excess gain of 2 kg in a year, representing an excess of 33 kcal per day.

A number of reports have presented evidence that obese children and adolescents are physically less active than non-obese children and

adolescents (Bruch, 1940; Johnson et al., 1956; Stefanik et al., 1959; Stunkard and Pestka, 1962; Bullen et al., 1964). However, whether the inactivity is a cause or a consequence of the obesity or is merely a characteristic of the obese individual is unknown. Neither do we know whether obese children actually expend less energy than do their non-obese peers. Bradfield et al. (1971) found no difference in energy expenditure between obese and non-obese high school girls. For performance of similar physical tasks, greater energy expenditures are required by obese than by normal subjects. Nevertheless, it is likely that decreased activity of obese subjects results in lesser overall energy expenditures than those of non-obese persons.

Whether obese children ingest more energy than do non-obese children is a question even more difficult to answer. Johnson et al. (1956) and Stefanik et al. (1959), using a diet history method, concluded that obese girls and boys consumed significantly less energy than did age-matched controls whereas Bradfield et al. (1971) reported opposite findings for a group of high school girls. As Garrow (1974b) has pointed out, variation of energy intake among normal subjects is so wide that small differences such as might exist between obese and non-obese subjects are unlikely to be detected.

It is apparent that the exact nature of the energy imbalance in obesity is unknown and it is possible that, at least in some instances, overeating may not be the most important reason for *remaining* obese. Nevertheless, it is quite likely that overeating is the major cause of *becoming* obese.

Consumption of an amount of food that is barely adequate for one child may represent overeating for another. Not only do energy expenditures at rest vary from individual to individual but activity patterns are also widely different. There is reason to believe that highly active children may be at relatively low risk of developing obesity. In a society in which food is readily available, nearly all children are likely to eat at times for reasons other than satisfying energy requirements—because of social pressures or merely because irresistibly attractive food is at hand. Under these circumstances, the child with high activity and consequent large energy expenditures seems likely to be at less risk from overeating than is the inactive child whose energy needs are readily met.

Although convincing studies of human subjects have not yet been reported (and are exceedingly difficult to carry out), a large body of evidence from animal studies (Fomon, 1974c) suggests that food consumed in one or two large meals is more conducive to development of obesity than is the same amount and type of food consumed in a number of small meals. The practice of many children (and adults) of obtaining a large percentage of calories at a single meal, often an extended evening meal, seems unwise.

Establishing Habits

It is likely that a large percentage of the population harbors the latent or overt inclination to overeat in the presence of an abundance of food. Unfortunately we do not yet know what allows one person to cope effectively with this constant opportunity to overeat while another person is unsuccessful and becomes obese.

Eating habits that encourage a relaxed attitude and acceptance of the fact that complete satiety cannot be maintained at all times will be most helpful. The earlier in life a habit—good or bad—is established, the more likely it is to persist; hence, the need to begin preventive efforts in infancy.

Bottle-feeding appears to promote more rapid gain in weight than does breast feeding (Stewart and Westropp, 1953; Mellander et al., 1959; Fomon et al., 1971), possibly reflecting a tendency of parents to overfeed when feeding is by bottle. It has been suggested (Fomon, 1971) that the breast-fed infant is permitted to stop nursing when he feels that he has had enough, whereas the bottle-fed infant is commonly encouraged to finish the last drop in the bottle. Once beikost [11] is introduced, the infant is likely to be encouraged not only to finish the last drop in the bottle but to finish the last spoonful in the dish. Such encouragement to overeat, feeding after feeding and day after day, might contribute to establishment of habits of overeating that would persist into later childhood and adulthood.

Because of the possibility that overfeeding during infancy and early childhood may lead to increased adipose tissue cellularity and/or may lead to establishment of habits of overeating, infant feeding practices merit reconsideration. With proper education, parents may be led to understand that a "good eater" is not a big eater but a moderate eater. From the first feedings, an infant should be permitted to stop nursing at breast or bottle at the earliest indication of satisfaction. The amount of formula in a feeding bottle should not be permitted to establish a goal for quantity to be consumed. With few exceptions, introduction of beikost should be discouraged until at least three months of age;[12] the amount in the serving dish should not be looked on as a goal for quantity to be consumed.

It seems unlikely that we shall be successful in teaching children habits of eating in moderation unless we are able to change prevailing attitudes of adults. With millions of persons in the world starving, it is certainly wrong to waste foods—as parents are quick to point out to

[11] Beikost is defined as foods other than milk or formula fed to infants.

[12] Although beikost is currently introduced during the first month, there is no evidence that this practice is beneficial. There is, in fact, little medical indication for introduction of beikost before five months of age. Three months represents a compromise.

their children. Yet, the major steps in avoiding waste must be sought in purchase of food, its preparation and serving. If a serving for a child exceeds his needs and yet is consumed entirely, the food is in a sense still wasted and, in addition, may contribute to development of obesity. To avoid waste, the emphasis must be on serving size rather than on eating all that is served.

It is well recognized that food is often used in expressing affection and emotions. The advice of Bruch (1973b) is worth emphasizing: "A mother must learn to differentiate between expressions of nutritional need and other discomforts, and she must not use food as a universal pacifier, or as reward, or withhold it as punishment."

Summary

A number of measures may be suggested for prevention of obesity: (1) Parents should be educated about the dangers of overfeeding and misuses of food during infancy and early childhood because of the possibility that habits of overeating may be established and persist into childhood and adult life. (2) Breast feeding should be encouraged and age of introduction of beikost should be delayed at least until 3 months of age. (3) Vigorous physical activity should be encouraged on a regular basis. (4) Efforts to develop appropriate community facilities for year-round physical activity of children (and adults) are important. (5) Several smaller meals are probably more effective in preventing obesity than one or two large meals providing the same energy intake. (6) When one or both parents are obese, the child is at greater risk of obesity than are most other children. Therefore, children from families with an obese parent should be identified and given particular guidance with respect to avoidance of excess weight gain. As stated by Bruch (1973c), ". . . . the existence of hereditary factors is often interpreted as condemning one to inactivity, that nothing can be done. Actually, obesity in other members of a family should be a call for greater alertness to prevent the potential of these inherited capacities from becoming a reality by avoiding the environmental influences which would encourage them to flourish. If proper limits are set a child so endowed may fulfill his potential for growth without becoming manifestly obese."

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CHAPTER 2
DIET AND ATHEROSCLEROSIS

SAMUEL J. FOMON

Deaths caused by myocardial infarction and stroke are more numerous than the next two leading causes, cancer and accident, combined (Killip, 1975). Myocardial infarction and stroke are consequences of atherosclerosis and atherosclerosis begins in infancy or childhood. The discussion that follows will attempt to review the current controversy regarding the desirability of introduction during childhood of measures aimed at prevention of atherosclerosis.

Definition, Pathogenesis and General Considerations

Atherosclerosis is a slowly progressive disease characterized by the formation of atheromata in the aorta and in medium and large arteries. The basic lesion—the atheroma—consists of a raised focal fibrofatty plaque within the intima, having a core of lipid (mainly cholesterol, usually complexed to proteins, and cholesterol esters) and a covering fibrous plaque (Robbins, 1974). While there is some argument about the nature of the initial step, the largest body of evidence supports the view that excessive infiltration of lipids or lipoproteins from the serum into the intima is the basic cause of development of atheromata (Robbins, 1974). It seems probable that the infiltration of lipids or lipoproteins (notably, cholesterol) into the intima of the artery is dependent on the concentrations of these substances in the plasma (Connor and Connor, 1972).

General Considerations

Myocardial infarction and stroke are almost certainly multifactorial conditions whose probability is controlled by genetic, metabolic, anatomic and environmental factors. It is estimated that only a small minority (perhaps less than 5%) of Americans have severe hyperlipidemia which is primarily based on genetic and not on environmental factors (Connor and Connor, 1972). Among children so affected, the great majority have familial hypercholesterolemia—the so-called Type II hyperlipoproteinemia (Fredrickson, 1972). Although this disorder is primarily genetic in origin, dietary therapy is considered an essential part of any program of management.

Among adults and probably among children, it seems likely that elevated serum concentrations of cholesterol more commonly result from environmental influences than from genetic factors. The greatest controversy about management concerns this group.

The Current Controversy

When measures aimed at preventing a major disease are of proven effectiveness, free of hazard, and offer little inconvenience, support of the great majority of health professionals may be anticipated. In the case of dietary measures aimed at prevention of atherosclerosis, effectiveness of the proposed intervention is somewhat uncertain, vague possibilities of risk may be suggested—at least for some age groups—and with foods currently available, inconvenience is considerable. Unfortunately, the balance of risk-benefit (or inconvenience-benefit) must be judged from somewhat inadequate data and it is therefore not surprising that there is lack of agreement on the desirability of introducing changes in diet.

Even among those who conclude that certain dietary restrictions should be broadly promoted for adults, agreement has not been reached about measures to be employed for infants and children. Measures defensible for adults may be considered inappropriate for infants and children because the threat that atherosclerosis presents to these age groups is several decades removed in time, because preventive measures instituted in early adult life may be fully adequate and because risks of intervention may be greater for younger individuals than for adults.

Some evidence of atherosclerosis has been reported in at least 45% of American men less than 30 years of age killed in wartime (Rigal et al., 1960; Mason, 1963; McNamara et al., 1971). In the study by McNamara et al. (1971) 5% were reported to have evidence of severe coronary atherosclerosis. It is therefore apparent that atherosclerotic lesions are present in young adult males. The cross-sectional studies of Holman et al. (1958) and of Strong and McGill (1969) suggest that atherosclerotic changes begin in infancy and progress throughout the life span. Children with cystic fibrosis of the pancreas are an exception. Such patients fail to absorb a major portion of dietary cholesterol and longchain saturated fatty acids and demonstrate relatively low serum concentrations of cholesterol (Luzzati and Hansen, 1944); at death their aortas exhibit much less atherosclerotic change than do those of children dying of other diseases (Holman et al., 1959).

Available evidence therefore strongly favors the view that atherosclerosis has its origins in childhood. Whether dietary restrictions should be widely promoted for children remains a matter of vigorous controversy.

The major controversy concerning prevention of atherosclerosis in the pediatric age group concerns dietary management of children be-

yond infancy who are not known to have familial hyperlipoproteinemia. Nondietary management (avoidance of smoking, encouragement of exercise) seem to be looked on with favor whether in relation to adults or children. For infants without familial hyperlipoproteinemia, individual authors and expert committees generally agree that dietary restriction of saturated fatty acids and cholesterol is not warranted.[13] Because human milk is a relatively rich source of cholesterol, any general recommendation for low intakes of cholesterol would amount to a repudiation of breast feeding. For children with familial hyperlipoproteinemia, individual authors and expert committees generally agree that dietary restriction of saturated fats and cholesterol is desirable.

The lack of agreement regarding the desirability of major modification of the diet of American children does not seem to reflect lack of agreement about whether atherosclerosis has its origins in childhood. As already noted, the evidence that atherosclerosis begins in childhood is reasonably convincing. The lack of agreement about the desirability of modifying children's diets arises because of (1) the uncertainty that modification will exert an appreciable beneficial effect and (2) the possibility that harm may result. Thoughtful opinions have been expressed on both sides of this issue. A number of them seem particularly worth citing.

Opinions Opposing Change in Children's Diets

A Subcommittee on Atherosclerosis, Council of Rheumatic Fever and Congenital Heart Disease of the American Heart Association (Mitchell et al., 1972) concluded that

> *. . . on the basis of data available there is no scientific justification at this time for recommending to the population at large that diets of all children be radically altered in the hope of preventing premature heart disease. However, the child at high risk (in particular Type II hyperlipoproteinemia) can be identified and should be placed on an appropriate diet.*

The Committee on Nutrition of the American Academy of Pediatrics (1972) came to a similar conclusion:

> *. . . dietary intervention should, before being recommended for everyone, be tested in persons with exceptionally high risk of having coronary heart disease—namely, those with familial type II hyperlipoproteinemia. Identifying these individuals early in life is feasible, and motivation for dietary restriction is likely to be greater in affected families than in the general population. The benefits, or possible adverse effects, of dietary changes can be evaluated from a study of this special group before changes are recommended for the*

[13] However, it may be noted in passing that most young infants in the United States receive commercially prepared formulas low in saturated fats and cholesterol.

population at large. A nationwide alteration in diet may well impair the sense of well-being of the general public, apart from any unforeseen, organic harm.

Schubert (1973) has expressed a similar opinion:

Eating should be a pleasurable experience but the development of good eating habits is difficult to achieve. American children as well as their parents like to eat steak, hamburger, hot dogs, sausage, eggs and ice cream. Although limitation of intake to prevent obesity is reasonable, restricting these foods in everyone for the unproved distant goal of reducing atherosclerosis is dubious . . .

McMillan (1973), although believing that dietary intervention is warranted in adult life, does not consider that such intervention is desirable in childhood:

Obviously there are serious deficits in our understanding of how atherosclerosis develops and what the mechanisms of its prevention may be. Consequently, several hypotheses can fit the available data. Nevertheless, for the general population, the critical time of life for atherogenesis and for prevention of future clinically significant disease appears to be in the young adult years and thereafter.

This conclusion should not be applied to that segment of the population who have especial liability to atherosclerosis. Their premature liability to severe lesions shifts the critical phase of atherogenesis back into the pediatric years.

Although not specifically opposing dietary intervention, several authors have emphasized the lack of proof of effectiveness of such change. One such opinion (Editorial, 1974) has been expressed as follows:

. . . So far, despite all the effort and money that has been spent, the evidence that eliminating risk factors will eliminate heart disease adds up to little more than zero in terms of preventing heart disease on a public health scale . . . Serum-cholesterol correlates positively with the incidence of events linked with atherosclerosis. Cause and effect is inferred, but as yet unproven on a large scale. A key test is to show that electively reducing serum-cholesterol reduces clinical atherosclerotic cardiovascular disease. The evidence in man is hopeful, but miniscule in comparison with the general aura of faith in such therapy.

Along a somewhat similar line of thought is the view of Corday and Corday (1975):

It appears that our nation's overconfidence in present risk factor concepts is impeding development of other promising preventive approaches.

These statements by responsible committees and investigators call attention to two important considerations: (1) It is not proven that

dietary intervention will prevent the clinical events associated with atherosclerosis and (2) one must consider the possibility that some risks may arise from dietary measures introduced during childhood.

A further point is worth noting. If dietary intervention should be shown to provide some benefit when a program is adhered to by adults, it would not necessarily be true that medically significant additional benefit would result from instituting such a dietary program during childhood.

Opinions Favoring Change in Children's Diets

As early as 1970, the Inter-Society Commission on Heart Disease Resources presented arguments favoring change in the national diet, including that of children. The recommended dietary modifications included adjustment of caloric intake to achieve optimal weight, reduction of dietary cholesterol to less than 300 mg per day and substantial reduction of dietary saturated fats. This Commission is currently reviewing its recommendations and a further report is anticipated.

Drash (1972) also has suggested that some change in eating habits may be necessary.

Should the eating habits of American children be changed? Much controversy surrounds the question of relationship between food ingestion and coronary artery disease, but accumulating evidence suggests that major alterations in the types and quantity of food ingested may be required in order to reduce the high mortality rate from atherosclerosis in our society.

The American pediatrician, traditionally concerned about nutrition, must accept an active role in determining what nutritional factors during childhood may contribute to the development of coronary artery disease. The success of any major effort to alter eating habits of the American public will depend largely upon the informed advice of the family pediatrician.

Blumenthal (1973) considers the evidence sufficient to warrant intervention in childhood.

The morbidity and mortality from atherosclerosis have reached such proportions that it has become one of the major challenges to medical science and public health in the United States . . .

Atherosclerosis appears to begin in childhood . . . There is little evidence that advanced lesions regress. Until such evidence is available, primary prevention will be essential, especially for those whose initial symptom of the disease is a fatal event. If it is true that advanced lesions are not apt to regress, prevention should be instituted early in life.

Kannel (1974) has offered a reasonable resolution to the current controversy.

> *Until more data on the efficacy of preventive measures becomes available, the practicing physician must decide for himself whether sufficient rationale for intervention exists. He must weigh the hazards against the potential benefits . . . If he elects to proceed on the incomplete evidence available, he can take comfort in the fact that the measures advocated are good health practices and have other benefits.*

Potential Hazards of Altering Diets of Infants and Children

The major question that the practitioner will need to have answered is, are there significant hazards? Concerns about safety of diets low in cholesterol and in saturated fatty acids for infants and children appear to be based on several possibilities: (1) that cholesterol may be an essential nutrient during early life—i.e., the requirement is greater than can be met entirely by endogenous synthesis—or that it may be a beneficial dietary component for other resasons; (2) that a major decrease in cholesterol-containing components of the diet will result in deficiencies of various essential nutrients; (3) that adverse consequences not yet identified will result from the alteration of the diet. Each of these concerns will be discussed.

Cholesterol is a precursor of bile acids and of steroid hormones and is a component of myelin, of cell membrane lipid and of the lipid of many organs. Even during infancy (a period when restricted intake of cholesterol has not been recommended), there is no evidence that bile acid formation is significantly influenced by cholesterol intake. Infants fed cholesterol-free formulas generally digest quite well the mixture of vegetable oils included in those formulas (Fomon, 1974a). For children beyond infancy, the possibility that restriction of cholesterol intake will reduce the size of the bile acid pool seems highly improbable. The data of Plotz et al. (1968) indicate that in the brain of the human fetus cholesterol is synthesized in situ from glucose and one presumes that after birth exogenous cholesterol is also of little importance in myelination.

The other basis for the belief that intake of dietary cholesterol may be desirable in early life relates to the establishment of appropriate feedback controls of cholesterol metabolism. Several authors (Hahn and Koldovsky, 1966; Fomon, 1971; Reiser and Sidelman, 1972) have suggested that an exogenous source of cholesterol may be desirable for the human infant. It is conceivable that dietary cholesterol is necessary during early infancy to induce enzyme systems responsible for normal functioning of the feedback mechanism regulating biosynthesis and catabolism (and/or excretion) of cholesterol. Such an adaptive change occurring during early postnatal life would not be unprecedented. Hahn

and Koldovsky (1966) have provided several examples of adaptive changes in infant animals. Studies of interest with respect to the possibility that such an adaptive change might occur in cholesterol metabolism have been reviewed (Fomon, 1974b). The results are compatible with the hypothesis that for male (but perhaps not for female) rats moderate rather than low intakes of cholesterol during the suckling period are conducive to development of satisfactory regulatory mechanisms for cholesterol metabolism in the adult. The studies have concerned relatively few animals and merit repetition both in the rat and in other species.

If cholesterol is an essential nutrient for the infant, it must be emphasized that the signs of deficiency are not readily apparent. In the United States, the three most widely fed commercially prepared infant formulas are quite low in cholesterol content and two of the three are nearly free of cholesterol. Many infants receiving these formulas do not consume other cholesterol-containing foods during the early months of life and, yet, no overt evidences of deficiency have been recognized.

In any case, it has been noted (Fomon, 1974c) that no official group has yet recommended restriction of dietary intake of cholesterol and saturated fatty acids of normal infants. The controversy concerns children more than 1 or 2 years of age. For this age group there is little reason to suspect that dietary intake of cholesterol is desirable.

The argument that a diet restricted in cholesterol will result in substantial decreases in intakes of milk, eggs, meat and cheese with consequent deficiencies in intakes of protein and trace minerals is unconvincing. Skim milk would not be restricted and lean meat would be permitted in moderation. Protein intakes in the United States are generally considerably greater than recommendations (Center for Disease Control, 1972; Owen et al., 1974; Fomon, 1974d) and some reduction in protein intake resulting from restriction in intake of eggs and dairy products rich in butterfat would be of little consequence. Neither is there reason to believe that trace mineral deficiency would be likely to develop from a diet restricted in cholesterol-rich foods.

There remains the vague uneasiness that any proposed dietary change may be followed by adverse consequences not yet identified. This possibility can neither be substantiated nor dismissed. Presumably, the physician must make his decision about the advisability of changing dietary habits by weighing this vague and unknown hazard, together with the inconvenience of the diet, against its possible but uncertain beneficial effects. The author's opinion is that benefits are likely to outweigh risks and that the inconvenience of the diet will not be so great as to prohibit change.

Familial Hyperlipoproteinemia

From the opinions cited previously, it is evident that in spite of the considerable controversy about the desirability of modifying the American diet, especially the diet of children, there is general agreement that dietary intervention is desirable in the case of children with type II hyperlipoproteinemia. As mentioned in Part I (p. 50), a serum concentration of cholesterol persistently greater than 230 mg/100 ml should be considered distinctly abnormal and indicates the need for further study and dietary intervention. Individuals with the heterozygous form of type II hyperlipoproteinemia have a greatly increased risk of early onset of ischemic heart disease (Slack, 1969; Kwiterovich et al., 1974).

There is no direct evidence indicating that strict adherence to a dietary regimen will alter the course of the disease. Such evidence will be difficult to obtain, as is the case with any disease for which the manifestations develop slowly over many years.

Dietary management alone will result in some decrease in serum concentration of cholesterol in patients with type II hyperlipoproteinemia and further decrease may be brought about by administration of hypocholesterolemic agents (Segall et al., 1970). A decrease in serum concentration of cholesterol *may* be associated with retardation in the progression of atherosclerotic changes. Convincing evidence of benefit is not available.

Dietary intervention methods consist of (1) measures aimed at weight control (see p. 69), (2) restriction in total fat intake with primary attention to restriction of intake of saturated fatty acids, (3) restriction of cholesterol intake.

Infants

In the case of infants, a diet low in content of cholesterol and saturated fatty acids presents little inconvenience. During the early months of life, a high percentage of normal infants receive commercially prepared formulas and most such formulas are nearly free of cholesterol and relatively low in content of saturated fatty acids. Little inconvenience is therefore encountered in providing a diet low in cholesterol and saturated fatty acids throughout the first year (and even throughout the first two years of life).

During infancy it is probably desirable to rely on dietary management alone even though this may be somewhat less effective than the combination of dietary management and administration of hypocholesterolemic agents.

The diet of infants who are not breastfed should consist of a commercially prepared infant formula with fat provided in the form of corn, soy or other vegetable oil not rich in saturated fatty acids. These formulas are nearly free of cholesterol. Fat should account for 40 to 50%

of caloric intake during the first 6 months of life and for 30 to 40% of caloric intake thereafter.

Children

For individuals beyond two years of age, dietary management is considerably more difficult. It is evident that the diet of the entire family, and not just that of the child with hypercholesterolemia, must be altered. In addition, even during the preschool years a considerable amount of education will be necessary to prepare the child to cope with situations of eating outside the home.

The major dietary restriction concerns fat of animal origin, with particular attention to eggs, butterfat (including that in cream, whole milk, ice cream and cheese), shellfish and fatty meats. Lean meats, fish, white meat of fowl (without the skin) should be eaten in moderation. Other foods are not restricted.

Summary

The major controversy concerning prevention of atherosclerosis in the pediatric age group concerns dietary management of children beyond infancy who are not known to have familial hyperlipoproteinemia. There appears to be general agreement that dietary intake of total fat, saturated fatty acids and cholesterol should not be restricted in normal infants, and many authors have noted that human milk is a rich source of these substances. It is also generally agreed that diets of children with familial hyperlipoproteinemia should be restricted in total fat, saturated fatty acids and cholesterol.

The lack of agreement about the desirability of modifying the diets of normal children beyond infancy does not seem to reflect lack of agreement about whether atherosclerosis has its origins in childhood. The evidence strongly suggests that it does. The question about desirability of dietary intervention arises because of (1) the uncertainty that modification will exert appreciable beneficial effect and (2) the possibility that harm may result.

The possibility of harm from a modified diet appears slight. However, as pointed out by Kannel (1974), for some time to come the judgment about the benefit-risk ratio will need to be made by the individual physician with the understanding of the patient and the family.

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CHAPTER 3
PREVENTION OF DENTAL CARIES

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As summarized by Scherp (1971), dental caries consists of localized, progressive decay of the teeth, initiated by demineralization of the outer surface of the tooth. This demineralization is caused by organic acids produced locally by bacteria that ferment deposits of dietary carbohydrates. Required for production of dental caries are the presence of a susceptible tooth, an oral environment conducive to the abundant growth and implantation of cariogenic microflora and a diet providing adequate substrate for the microorganisms. Dental caries is usually a chronic disease requiring considerable time for the destructive process to become clinically evident.

There can be little question of the high prevalence of dental caries. In the Preschool Nutrition Survey, Owen et al. (1974) reported an average of 2.6 decayed, extracted or filled deciduous teeth in white children and 3.8 such teeth in black children between 4 and 5 years of age. Findings of the Ten-State Nutrition Survey (Center for Disease Control, 1972) indicate that dental caries is even more prevalent as age increases. Corresponding averages for children between 5 and 6 years of age were 3.7 and 5.1. By age 10, more than 80 percent of children have caries of permanent teeth (Downer, 1970; Palmer, 1971). In a random sample of nearly 5000 Navy recruits, only one was found to be caries-free (Newbrun, 1969). The Committee on Nutrition (1972) concluded that dental caries is the most prevalent disease for all age groups beyond infancy.

Foods and Feeding

Carbohydrates

Various carbohydrates serve as substrate for the microflora of the mouth. Cariogenic streptococci, such as Streptococcus mutans, anaerobically metabolize sucrose to glucose and fructose (or metabolize other simple carbohydrates to their monosaccharide components), from which long chain polymers are formed—dextrans or glucans from glucose, and levans or fructans from fructose (Mäkinen, 1972). The dextrans and levans are major contributors to formation of plaques which adhere to the teeth as sticky, gelatinous masses. Because they resemble the tooth

in color and are somewhat translucent, plaques are difficult to see unless they are stained with erythrocin, fast green or a similar dye. Lactic acid and other organic acids produced by the bacteria beneath a plaque are protected from the buffering effect of saliva and are therefore able to bring about demineralization of the enamel. Thus, the primary substrate for the production of plaque and of organic acids is refined carbohydrates, especially sugars. Starches are less effective substrates because of their relatively slow fermentation in the mouth.

The majority of investigators appear to have concluded that sucrose is the most cariogenic dietary carbohydrate (Newbrun, 1969; Brown, 1975). The cariogenicity of sucrose has been demonstrated in vitro and in studies of experimental animals. Rather convincing circumstantial evidence implicates sucrose as a causative agent of dental caries in human subjects. First, epidemiologic studies (Sognnaes, 1948, 1949; Toverud, 1949; Toverud et al., 1961) have demonstrated a significant decrease in the dental caries rate among school children in occupied countries during the second half of World War II and the return of the caries rate to prewar levels after the war. These changes in caries rate coincided with the wide availability of sucrose before and after the war and its relative unavailability during the second half of the war. Second, in a study conducted under controlled conditions in an institution (Gustafsson et al., 1954), frequent and long-term consumption of sticky candy was associated with increase in the dental caries rate. In Australia, children who had lived from infancy to puberty in Hopewood House, where a lacto-vegetarian diet with almost complete absence of refined carbohydrate was consumed, demonstrated significantly fewer decayed, missing and filled teeth than did children living under similar socioeconomic conditions in state schools but not adhering to the same dietary regimen (Harris, 1963). In a study of preschool children, frequency of consumption of between-meal snacks appeared to be correlated with dental decay rate (Weiss and Trithart, 1960). Finally, patients with the rare metabolic disorder, hereditary fructose intolerance, who develop nausea and vomiting when they consume fructose (including the fructose in sucrose), learn to avoid fructose-containing foods; these subjects are caries free or have extremely low dental caries rate (Newbrun, 1969; Scherp, 1971).

Nursing Bottle Caries

"Nursing bottle caries" refers to destruction of the anterior teeth as the result of prolonged contact of the teeth with carbohydrate-containing solutions fed by nursing bottle. When an infant is given a sugar-containing fluid as a pacifier, especially at bedtime or naptime, sucking and swallowing is infrequent, saliva flow is minimal and the sugar remains in contact with the teeth for a long time. Most seriously affected are the anterior maxillary teeth, particularly the interproximal and labial surfaces (Fig. 3–1). Sometimes the entire crowns are destroyed (Fig. 3–2).

Figure 3-1 Nursing bottle caries affecting the maxillary anterior teeth and the first primary molars in an 18-month old child. Note the extensive destruction of the labial surfaces of maxillary incisors (arrow) and the occlusal surfaces of the first primary molars. The lingual surfaces of the maxillary incisors (not shown) are also severely decayed.

Figure 3-2 A typical example of a child affected with nursing bottle caries. Note the total destruction of the maxillary incisors. The first primary molars have required the restoration with stainless steel crowns. In the early stages, the lower teeth are relatively unaffected.

Usually, the mandibular incisors, which are protected by the tongue and submaxillary salivary secretions, remain free of caries.

The lactose naturally present in cow milk is probably not a major threat with respect to nursing bottle caries. Of more concern is the content of sucrose and corn syrup solids in a number of infant formulas, especially soy isolate-based and other milk-free formulas. Probably most threatening is the feeding of sweetened fruit-flavored drinks and sweetened fruit juices. It seems undesirable to feed sweetened fluids (including formula) by bottle once the teeth have erupted. The practice of permitting infants to use a bottle as a pacifier, especially at bedtime, should be discouraged.

Other Dietary Considerations

Various reports in the literature suggest that certain foods, such as cow milk, cheese and fibrous foods (e.g., celery, carrot) may exert a protective effect against the action of sucrose and other cariogenic substances in the diet (Caldwell, 1970; Wei, 1974a). Apples and other fruits contain fructose, a less cariogenic sugar than sucrose. Although excessive exposure to fructose may produce dental caries, fresh fruits are likely to be much less cariogenic than most sucrose rich snack foods consumed by small children. Phytates and phosphates (both organic and inorganic) may also exert a protective effect against dental caries. However, thus far, most studies have been conducted in vitro or with the rat as the experimental animal. There is not yet convincing evidence that in the human any foods or food components other than fluoride are protective against cariogenic substances in the diet. This is an important area for further study.

Eating Between Meals

Because refined carbohydrates exert their effect in promoting dental caries by serving as a substrate for caries-producing streptococci, it is apparent that for older children as well as for infants not only the total quantity but the form of the carbohydrate and the frequency of consumption are important. A single piece of sticky candy may adhere to the teeth for almost an hour. In the case of sugars that are not in sticky form, a specified amount consumed at one time is likely to be less conducive to formation of dental caries than the same amount consumed in small portions throughout the day.

Considerable evidence exists that between-meal snacks favor development of dental caries (Zita et al., 1959; Weiss and Trithart, 1960). Presumably, it is primarily the simple sugars that are responsible. In the case of schoolage children, it may be feasible to reduce or eliminate eating between meals, although a number of studies indicate that large, in-

frequent meals may predispose to obesity and have other adverse effects (Fomon, 1974).

It would seem to be a sound policy to recommend that school canteens and concession stands refrain from selling sweets. Roder (1973) concluded that the motivation for selling sweets in school canteens and concession stands related to the belief that such sale was necessary in order to make a profit. It is rationalized that sweets sold in schools comprised a relatively small percentage of total daily carbohydrate intake and therefore were unlikely to be an important factor in promoting dental caries. However, the study showed a positive correlation between the availability of sweets in school canteens and the dental decay rate in primary and secondary school children. Furthermore, the study showed that canteens omitting sweets can be highly profitable.

Perhaps it is self-evident that a diet suitable for preventing one disease may contribute to development of another. Health workers need to take a broad rather than a narrow view of preventive medicine. Those dedicated to research and teaching in prevention of iron deficiency, obesity, atherosclerosis and other diseases should work jointly with those active in research and teaching in preventive dentistry. Adjustment of the diet for the purpose of prevention of dental caries should not increase the risk of other disorders.

It is evident that forbidding children to eat between meals is likely to meet with quite limited success. A better approach may be development of a list of foods to be avoided between meals and another list of foods to be permitted. Foods to be avoided are the following: sugar, honey, corn syrup, candies, jellies, jams, sugared breakfast cereals, cookies, cakes, chewing gum and sweetened beverages, including flavored milks, carbonated drinks, sweetened fruit juices and fruit or fruit-flavored drinks.

As discussed previously (p. 79), a decision may be made jointly by some families and their physicians to restrict intake of cholesterol and saturated fatty acids as a preventive measure against subsequent development of atherosclerosis. Between-meal snacks for children in such families may include fresh fruits and vegetables, breads and crackers with margarine or peanut butter, low fat (or filled milk) cheeses, lean meats and "2 percent" or skim milk. Many other foods that are neither cariogenic nor atherogenic may be added to this list according to individual preferences.

Cleaning the Teeth

Parents should be instructed to begin cleaning the teeth of toddlers as soon as it is practical to do so. Fine gauze wrapped around the parent's finger is satisfactory at first. Brushing by the parent may be possible with some children even before 18 months of age. Toothpaste should be considered optional and its use should depend on acceptance by the child.

Fluoride

An adequate daily intake of fluoride may exert its beneficial effect in decreasing the incidence and severity of dental caries in a variety of ways (Wei, 1974b). However, the major effects appear to be (1) the conversion of the enamel mineral, hydroxyapatite, to fluorapatite with a consequent reduction in acid solubility (Newbrun, 1972), (2) promotion of remineralization and (3) antibacterial and anti-enzyme effects resulting in decreased acid production.

Fluoridation of Drinking Water

When fluoridated water (0.7 to 1.2 mg of fluoride per liter of water) is consumed throughout life, the dental caries attack rate is reduced by 50 to 60 percent in permanent teeth (Backer Dirks, 1974) and slightly less in primary teeth (Scherp, 1971). The most likely explanation for the lesser reduction in dental caries attack rate in primary than in secondary teeth is that many infants and small children consume relatively little drinking water—suggesting the desirability of administering fluoride supplements (see *Single Daily Dose of Fluoride*).

An excessive ingestion of fluoride causes mottled discoloration of the teeth. Moller (1965) indicated the degree of fluorosis using arbitrary scores of normal 0.0, questionable 0.5, very mild 1.0, mild 2.0, moderate 3.0, and severe 4.0. The categories of questionable and very mild fluorosis are cosmetically acceptable and are not noticeable by the layman. Moderate and severe fluorosis may acquire brown stains as a result of local oral factors and are cosmetically objectionable (Armstrong, 1972).

The relation between the dental caries experience and extent of fluorosis in various communities in relation to the fluoride content of drinking water is indicated in Figure 3–3. This figure concerns observations of the permanent teeth of 7000 children 12 to 14 years old who were lifelong residents of communities with the designated fluoride content and who had never left their communities for more than 1 month (Hodge, 1975). The average number of decayed, missing and filled teeth decreases from 8 at very low concentrations of fluoride in drinking water (0.1 ppm) to less than 3 when the fluoride content is approximately 1 ppm.

The index of fluorosis remains near zero with increasing fluoride concentrations in drinking water until concentrations reach values greater than 1.0 ppm. With further increases in fluoride concentration of the drinking water, the index of fluorosis rises steeply. In the communities of Galesburg and Elmhurst, Illinois (indicated by an arrow in the figure), the natural fluoride content of drinking water was 1.9 and 1.8 ppm, respectively, and the indices of fluorosis were 0.69 and 0.68, respectively. More than 1 percent of the 443 children in these communities

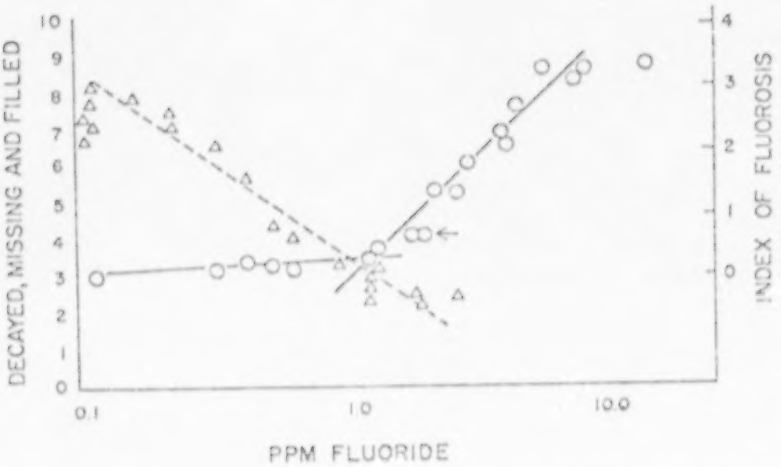

Figure 3-3 The relationship between fluoride content of drinking water, average number of decayed, missing and filled teeth (left-hand ordinate), and index of fluorosis (right-hand ordinate). Each triangle applies to observations concerning decayed, missing and filled teeth in children in one community. Each open circle concerns the index of fluorosis in one community. The arrow indicates the communities of Galesburg (left) and Elmhurst (right), which are mentioned in the text. (Modified from Hodge and Smith, 1954).

had moderate fluorosis. Small (1973) has concluded that communities where fluoride concentration of drinking water is 1.6 ppm or less may be expected to demonstrate acceptably low fluorosis indices (0.6 or less).

Of the total United States population of 213 million in 1975, approximately 177 million persons used public water supplies and about 105 million (59 percent) of these individuals received water supplies that were fluoridated at least to 0.7 ppm either naturally (5.0 percent of all persons) or artificially (44.4 percent) (Dental Disease Prevention Activity, 1977). Approximately 36 million persons live in rural areas without communal water supply and therefore cannot benefit from fluoridation. Increasingly it has been recognized that the benefits of water fluoridation are both systemic and topical and that the topical effect may be the more important. Small, frequent doses of topically applied fluoride appear more efficacious than yearly applications of high concentrations (Wei, 1974a). In Sweden the weekly use of sodium fluoride mouth rinses by school children was associated with reduction in caries rates comparable to those of communities with water fluoridation (Torell and Ericsson, 1974). Fluoride rinses are not recommended for preschool children, however, because many young children cannot control their swallowing reflexes and may swallow most of the rinse.

Single Daily Dose of Fluoride

Because of the large number of individuals who do not have access to fluoridated drinking water, the effectiveness of single daily doses of fluoride is a matter of considerable interest. There can be no doubt that single daily dosage of fluoride in drop or tablet form reduces the caries attack rate (O'Meara, 1968; Stookey, 1970; Hennon et al., 1972; Aasenden and Peebles, 1974; Driscoll, 1974). Combined fluoride-vitamin preparations appear to be more readily accepted than vitamin-free fluoride preparations (Arnold et al., 1960; Richardson, 1967) although, unfortunately, such preparations do not permit the desirable adjustment of fluoride dosage without adversely affecting the vitamin dosage. A large chewable tablet or lozenge will impart additional topical benefits to the teeth and is preferable to small tablets that are usually quickly swallowed.

When single dose fluoride supplements are administered, it is, of course, desirable to achieve maximal protection against dental caries with minimal risk of fluorosis of the enamel. Considerable uncertainty exists, particularly in the case of infants and toddlers, with respect to the dose most likely to accomplish this end (Ericsson and Ribelius, 1971). Newer information from several sources (Forrester and Schulz, 1974; Aasenden and Peebles, 1974; Kramer et al., 1974; Fanning et al., 1975; Infante, 1975; Wiatrowski et al., 1975) suggests that previous recommendations for fluoride supplementation of infants and small chil-

TABLE 3-1

RECOMMENDED FLUORIDE SUPPLEMENTATION

Fluoride Concentration of Water Supply (ppm)	Desirable Fluoride Supplementation (mg/day)				
	Age 0–6 months	*Age 6–18 months*	*Age 18–36 months*	*Age 3–6 years*	*Age 6 years*
0.2	0*	0.25	0.5	0.75	1.0
0.2–0.4	0*	0*	0.25	0.5	0.75
0.4–0.6	0*	0*	0	0.25	0.5
0.6–0.8	0*	0*	0	0	0.25
0.8	0*	0*	0	0	0**

*0.25 for fully breastfed infants.

**In this age group, the hazard of fluorosis is low and some additional protection will probably be afforded by fluoride supplementation. However, fluoride supplementation is probably not desirable when drinking water provides more than 1.1 ppm.

dren (Wei, 1974a; American Dental Association, 1975) may lead to somewhat greater intakes of fluoride than are desirable.

As will be discussed, no fluoride supplementation is recommended during the first 6 months of life, a maximum dosage of 0.25 mg daily between 6 and 18 months, 0.5 mg daily between 18 and 36 months and a maximum of 0.75 mg daily between 3 and 6 years of age (Table 3–1). It is anticipated that these recommendations may require modification as additional evidence becomes available.

BIRTH TO AGE SIX MONTHS:

It had previously been assumed (Wei, 1974a) that infants living in communities with non-fluoridated drinking water and those who are breastfed or fed whole cow milk would be likely to receive only a small fraction of a milligram of fluoride daily. However, more recent data suggest that many infants may receive more dietary fluoride than was previously thought (Wiatrowski et al., 1975). In addition, fluoride content of commercially prepared concentrated liquid formulas may vary widely even among products produced by the same manufacturer. A concentrated liquid formula made from liquid fat-free milk does not require addition of water during its manufacture and will have low fluoride content. However, formulas may be made from dry, fat-free milk solids, from dry, demineralized whey and other dry ingredients or from a variety of other dry components (which applies to soy isolate-based formulas and most other milk-free formulas). In the case of formulas made from dry components, the manufacturer adds water from local water supplies in preparing the concentrated liquid product. Because a manufacturer may market a product of the same name produced in plants in different cities, it is apparent that the physician may not know the fluoride content of the feeding he recommends.

Whether an appropriate single daily dose of fluoride beginning at birth confers significantly greater protection against dental caries than does an appropriate single daily dose of fluoride beginning at age 6 months is currently unknown. In view of this uncertainty and the extreme difficulty in defining an appropriate daily dose of fluoride for infants less than 6 months of age, it seems desirable at present to delay fluoride supplementation to age 6 months (Table 3–1).*

SIX MONTHS TO THREE YEARS:

Because of the possibility that infants may receive greater intakes of fluoride from various foods than was previously assumed (Wiatrowski et al., 1975), the fluoride dosage of 0.5 mg daily recommended previously (Wei, 1974a) may be too high. Aasenden and Peebles (1974) have shown that children living in a community with low fluoride con-

* The fully breastfed infant is an exception because intake of fluoride is uniformly low. Supplementation with 0.25 mg fluoride daily seems reasonable.

centration in drinking water who received a supplement of 0.5 mg of fluoride daily from birth to 3 years and 1 mg thereafter showed a significant caries reduction (80%) compared to the control, but 67% of the children were classified as having fluorosis, mainly of the very mild or mild types. Therefore, a maximal daily dose of 0.25 mg of fluoride is recommended for children between 6 and 18 months of age (Table 3–1).

CHILDREN OVER THREE YEARS OF AGE:

As indicated in Table 3–1, older preschool children and school-age children should also receive fluoride supplements in relation to the fluoride content of the drinking water.

A large number of studies have demonstrated that the supervised use of a sodium fluoride mouth rinse (0.025% daily or 0.2% at intervals of two weeks) at school or at home may produce cariostatic effects similar to, if not superior to, those of water fluoridation. This represents an economic and practical public health solution in non-fluoridated communities (Torrell and Ericsson, 1974). Mouth rinses are contraindicated in preschool children because of the inability of the latter to control the swallowing reflex with the possibility of excessive ingestion.

Establishing Habits

During early infancy, when other preventive measures are being undertaken (e.g., immunizations against communicable diseases), parents may be particularly receptive to measures aimed at prevention of dental caries. Ideally, periodic examinations and counseling by the dentist should be repeated at intervals of six months until adolescence.

Although it seems desirable to delay fluoride supplementation until 6 months of age, in areas in which water supplies are low in fluoride education of parents regarding the desirability of fluoride supplementation should be initiated well before the infant reaches 6 months of age.

Parents should be taught proper technics of cleaning the child's teeth (brushing is not synonymous with cleaning); proper cleaning should be initiated during the second year of life, always under parental supervision. Young preschool children are unable to brush their teeth adequately without supervision (McClure, 1966).

Eating habits are established in infancy and early childhood. As already mentioned, it does not seem desirable to restrict food intake of one- to three-year-old children to three meals daily. Rather, the choice of foods eaten between meals should be supervised, and these foods should contribute to an adequate intake of all desired nutrients. Intake of sucrose and other nutritive sugars should be restricted, especially between meals, and sticky candies should be avoided.

Health workers should join forces in their efforts at establishing habits conducive to disease prevention. Efforts aimed at prevention of

major nutritional disorders—dental caries, obesity, atherosclerosis and hypertension—should be coordinated for maximum effect and for avoidance of confusion of children and their parents.

Summary

Dental caries is a widespread disease with serious consequences—pain, expense and loss of teeth. It must be considered a nutritional disorder because dietary carbohydrates are essential in its production and because the nutrient, fluoride, is effective in reducing the incidence of the disorder.

Sucrose and other simple sugars provide substrate for oral bacteria that produce plaque and, beneath the plaque, organic acids that erode the enamel of the tooth. The frequency of consumption and physical form of such sugars is of prime importance. Frequent consumption of sticky sweets will put the individual at high risk of developing dental caries. Foods eaten between meals should be those of low cariogenicity (see p. 86).

Recent reports suggest that intakes of fluoride by infants are, at least in some instances, likely to be considerably greater than had previously been supposed. Because of this newer evidence and considerable uncertainty that will exist about actual intakes of fluoride by individual infants, supplementation with fluoride is not recommended before six months of age. This recommendation will need to be reevaluated if in the future it is shown that supplementation with fluoride beginning at birth provides significantly greater protection against dental caries than does supplementation begun at 6 months of age.

The importance of early establishment of sound habits is emphasized. In particular, these habits relate to cleaning the teeth, avoiding undue exposure to simple carbohydrates and maintaining desirable intakes of fluoride.

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CHAPTER 4
PREVENTION OF IRON-DEFICIENCY ANEMIA

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The substantial literature on iron-deficiency anemia in infants and children is difficult to interpret because of uncertainties about definitions, variable reports of prevalence in different age groups, lack of agreement about extent of absorption of various forms of iron and disagreements about dietary requirements. The discussion that follows concerns these topics.

Definitions

Anemia

It has been argued that a moderately decreased concentration of hemoglobin is of little consequence unless it causes decreased capacity for physical work by imposing an increased load on cardio-respiratory mechanisms (Elwood et al., 1968). As stated by Oski (1973), "Not until physicians begin to think of anemia as a physiologic or biochemical derangement rather than a mere reduction in red cell mass will they be able to answer the seemingly simple question, 'Is this patient anemic?' "

There is little evidence that symptoms are related to hemoglobin concentration when concentrations are greater than 8 gm/100 ml (Elwood, 1973). In the absence of a satisfactory physiologic or medical basis for defining anemia, an arbitrary definition must be used. From the practical point of view, such a definition should be based on a readily measurable parameter, such as hemoglobin concentration or hematocrit. It seems unreasonable to establish different arbitrary values for various subgroups based on age, sex or race unless strong theoretic support can be offered for the assumption that a specified value suitable for one group is unsuitable for another.

The definition of anemia employed in this publication is a minor modification of that proposed by the World Health Organization (WHO Group of Experts, 1972). Anemia is defined here as a condition in which concentration of hemoglobin and/or the hematocrit is less than the values indicated in Table 4–1 for individuals living at sea level.

These arbitarily selected lower limits of normal are generally consistent with reports in the literature pertaining to individuals presumed

TABLE 4–1

HEMOGLOBIN CONCENTRATIONS AND HEMATOCRITS BELOW WHICH ANEMIA IS ASSUMED TO BE PRESENT

Age (year)	Sex	Hemoglobin Concentration (gm/100 ml)	Hematocrit
½–10	both	11.0	34
10–14	both	12.0	37
14+	male	13.0	41
14+	female	12.0	37

or known to be in satisfactory to excellent iron nutritional status. This relationship is illustrated in Fig. 4–1 for data that permit calculation of a value 2 standard deviations below the mean (Moe, 1965; Natvig et al., 1967; Scott and Pritchard, 1967; Marner, 1969; Owen et al., 1971; Samuelson and Sjölin, 1972; Hunter and Smith, 1972). With few exceptions, the minus 2 standard deviation value for hemoglobin concentration falls either slightly above or less than 0.5 g/100 ml below the proposed lower limit of normal. Other reports (Sturgeon, 1958; Haddy et al., 1974) do not permit calculation of a value 2 standard deviations below the mean; however, the mean values in these studies are similar to those cited for the age group and it seems likely that the standard deviations may also be similar.

The lower limits of normal proposed here differ from those of the WHO Group of Experts (1972) only for children from 6 to 10 years of age. For these children, the WHO Scientific Group proposes a hemoglobin concentration of 12.0 g/100 ml as the lower limit of normal. As may be seen from Figure 4–1, the minus 2 standard deviation values of Natvig et al. (1967) for Norwegian children 7 to 9 years of age fall well below a value of 12.0 g/100 ml. In fact, for 7-year-old children the value corresponding to the mean minus 1 standard deviation is 11.7 g/100 ml. Yet, there is no reason to believe that the 7- to 9-year-old

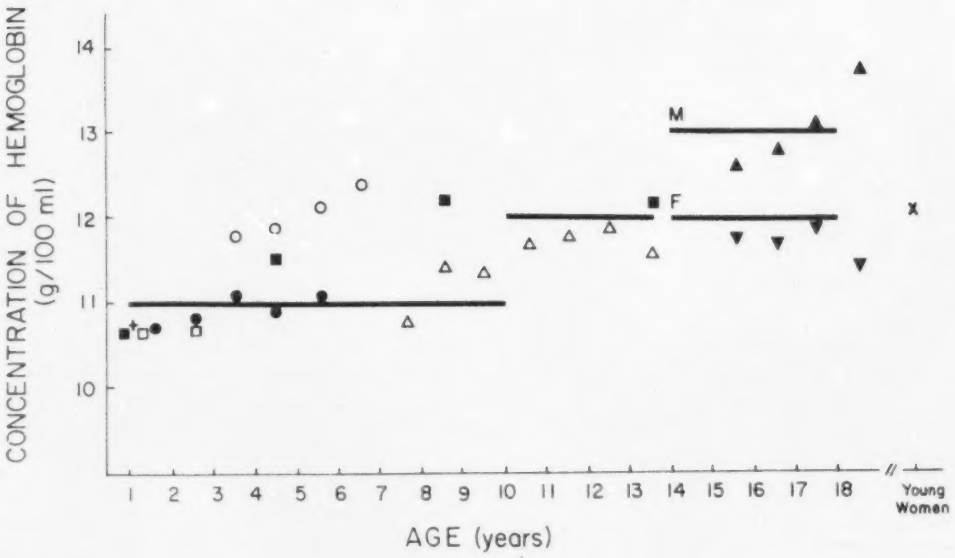

Figure 4–1 Data from literature plotted in relation to arbitrarily selected lower limits of normal (solid lines) for hemoglobin concentrations as indicated in Table 4–1. Each point indicates the value 2 standard deviations below the mean for a group of individuals believed to be in good or excellent iron nutritional status (see text): solid squares, Samuelson and Sjölin (1972); plus sign, children with saturation of transferrin greater than 16%, Hunter and Smith (1972); open squares, Moe (1965); solid circles, highest income group (Warner IV) studied by Owen et al. (1971); open circles, Marner (1969); open triangles, Natvig et al. (1967); solid upright triangles, males, Natvig et al. (1967); solid inverted triangles, females, Natvig et al. (1967); value for young women, Scott and Pritchard (1967).

children were in less satisfactory iron nutritional status than the older subjects studied by these same investigators.

Concentrations of hemoglobin less than 11.0 g/100 ml will be encountered relatively frequently in individuals less than 2 years of age. However, a high percentage of such infants and children will demonstrate low percent saturation of transferrin (Hunter and Smith, 1972; Haddy et al., 1974) and elevated ratio of free erythrocyte porphyrins to hemoglobin (Piomelli et al., 1976) (p. 49). There is, therefore, reason to believe that the lower limit of normal, even for the youngest age group, should not be less than 11.0 g/100 ml.

As discussed in Part I (p. 49), when hemoglobin concentrations are between 10.5 and 11.0 g/100 ml in individuals less than 2 years of age and current dietary intake of iron is adequate (p. 106), hematologic evaluation may not be necessary. However, such children should remain under closer medical supervision than those with greater concentrations of hemoglobin. Similarly, in the case of males between 14 and 16 years of age, if hemoglobin concentrations are between 12.0 and 13.0 g/100 ml, advice about dietary intake of iron and subsequent followup is likely to be adequate and hematologic investigation need not be undertaken.

The greater concentration of hemoglobin in males than in females after puberty is presumably related to greater circulating levels of androgens. The effect of androgens on hematopoiesis in experimental animals and human subjects is well recognized (Kennedy and Gilbertsen, 1957).

Because current definitions of anemia are arbitrary, it is to be anticipated that different values for hemoglobin concentration (or hematocrit) will be employed by various authors and committees. Such lack of uniformity is probably unavoidable until a definition of anemia can be based on acceptable physiologic or medical criteria.

Certain special circumstances warrant adjustment of the suggested values for hemoglobin concentration. Data from several sources (Albritton, 1952; Altman and Dittmer, 1961) suggest that hemoglobin concentrations may be approximately 1 gm/100 ml greater in adults living at an altitude of 1,500 meters (4800 ft) than in those living at sea level. Data of Owen (1976) from the National Preschool Nutrition Survey demonstrated that hemoglobin concentrations of white children 2 to 6 years of age averaged approximately 0.6 gm/100 ml greater for those living at an altitude of 1250 meters than for those living at lesser altitudes (the majority near sea level). More recently, Owen (1976) has found that the mean concentration of hemoglobin of iron-sufficient 4-year-old children living in Flagstaff, Arizona (2250 meters) is approximately 1 g/100 ml greater than the mean for this group in the National Preschool Nutrition Survey (Owen et al., 1974). Similarly,

hemoglobin concentrations of infants and children with cyanotic congenital heart disease are far in excess of those of normal children.

Iron-Deficiency Anemia

Iron-deficiency anemia is defined as a state in which the concentration of hemoglobin is below the limits of normal (see Anemia) and the existence of iron-deficiency is documented by one or more of the following: (1) reticulocyte response and/or increase in hemoglobin concentration after administration of iron, (2) ratio of free erythrocyte porphyrins (FEP) to hemoglobin in blood greater than 5.5 μg per gram (Piomelli et al., 1976), (3) saturation of transferrin in serum less than 16 or 17% (Hunter and Smith, 1972; Haddy et al., 1974), (4) serum concentration of ferritin less than 7 ng/ml (Siimes et al., 1974).

Circumstantial evidence from clinical studies suggests that the concentration of ferritin (the major form of storage iron) in plasma normally reflects the amount of ferritin in the reticulo-endothelial system (Jacobs and Worwood, 1975). There is a close quantitative relation between the two at all levels of iron storage from the deficient state to gross overload. In early infancy the shift of iron from the red cell to the storage compartment during the first six weeks of life is reflected by a sharp increase in concentration of ferritin in serum (Siimes et al., 1974; Rios et al., 1975). An equally sharp fall in serum concentration of ferritin occurs during the subsequent three months as storage iron is used for erythropoiesis. In iron-deficiency, the serum ferritin assay is usually diagnostic and therefore superior to determination of serum iron concentration or iron binding capacity (Siimes et al., 1974). Determination of serum concentration of ferritin is a promising approach to identification of iron-deficiency and it is to be anticipated that within a few years methods for determining ferritin concentration will become more widely available.

With the exception of infants less than 6 months of age, the younger the infant or child, the more likely it is that a low concentration of hemoglobin results from iron-deficiency.

Prevalence

In the United States and in most other countries, concentrations of hemoglobin below the normal range (Table 4–1) are seen most commonly in (1) infants of low birth weight, (2) full-size infants between 6 and 24 months of age, and (3) children and adolescents from low-income families.

Low-birth-weight infants

Since total body iron at birth is less in low-birth-weight infants than in term infants, low-birth-weight infants are at a markedly increased

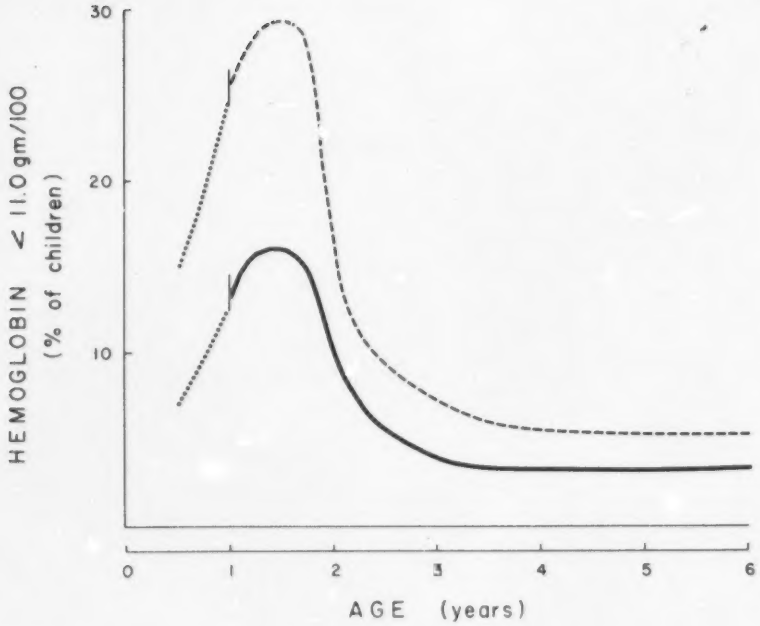

Figure 4–2 Percent of children with hemoglobin concentrations less than 11.0 gm/100 ml at various ages among the lowest income group (upper curve) and other income groups combined (lower curve). Data concerning one- to six-year-old children are from the National Preschool Nutrition Survey (Owen et al., 1974). Portions of curves for infants less than one year of age are estimated from other data (Fomon, 1974a).

risk of developing iron-deficiency anemia. Gorten and Cross (1964) reported that 25 of 76 low-birth-weight infants receiving a formula containing no added iron developed hemoglobin concentrations less han 9.0 g/100 ml before 18 months of age. None of 69 infants receiving a formula containing 12 mg per quart of elemental iron in the form of ferrous sulfate developed hemoglobin concentrations less than 9.0 g/100 ml.

Children Less Than 10 Years of Age

Figure 4–2 presents data of Owen et al. (1974) on percentage of children of various ages with concentrations of hemoglobin less than 11.0 g/100 ml. The difference in prevalence of low hemoglobin concentrations in relation to age and economic status is evident. A number of other studies of iron-deficiency anemia of infants and preschool children have been summarized by Fomon (1974a) and the findings seem generally in keeping with the data of Owen et al. (1974).

With the exception of the lowest income group, only 4% of children between 2 and 6 years of age in the Preschool Nutrition Survey (Owen

TABLE 4-2

SUMMARY OF LOW HEMOGLOBIN CONCENTRATIONS AND LOW HEMATOCRIT VALUES IN NATIONAL PRESCHOOL NUTRITION SURVEY (Owen et al., 1971)

Age* (years)	Lowest Income Group**				Other Income Groups			
	Hemoglobin		Hematocrit		Hemoglobin		Hematocrit	
	Number	Percent <11.0 gm/100 ml	Number	Percent <34	Number	Percent <11.0 gm/100 ml	Number	Percent <34
1-2	69	27	71	25	280	13	286	15
2-3	81	10	83	8	343	5	345	9
3-4	94	8	94	16	375	3	377	7
4-5	77	9	78	5	366	4	371	7
5-6	83	6	84	5	371	4	371	5
2-6	335	8	339	10	1455	4	1464	7

*Age 1-2 years indicates that child has reached first birthday and has not yet reached second birthday.
**Accounted for the lowest 18% of families by Warner classification.

et al., 1974) demonstrated concentrations of hemoglobin less than 11.0 g/100 ml (Table 4–2). Similarly, in the Health and Nutrition Examination Survey (Abraham et al., 1974), only 6.5% of children between 1 and 5 years of age living in families classified as above the poverty level demonstrated concentrations of hemoglobin less than 11.0 g/100 ml. Presumably, low hemoglobin concentrations were more prevalent in children between 1 and 2 years of age than in those between 2 and 5.

As already discussed (p. 97), in various studies of children less than 10 years of age and believed to be in good iron nutritional status, the concentration of hemoglobin corresponding to the mean minus 2 standard deviations is generally greater than 11.0 g/100 ml, although in a few instances the value falls between 10.5 and 11.0 g/100 ml (Fig. 4–1).

Data from a variety of sources indicate that the prevalence of low hemoglobin concentrations is much greater in the lowest income groups than in other income groups. As may be seen from Table 4–2, 9% of children between 2 and 6 years of age in the lowest income group of the Preschool Nutrition Survey demonstrated concentrations of hemoglobin less than 11.0 g/100 ml. In the Health and Nutrition Examination Survey (Abraham et al., 1974), hemoglobin concentrations were less than 11.0 g/100 ml in 13.8% of children between 1 and 5 years of age living in families classified as below the poverty level.

The prevalence of low hemoglobin concentrations and of low hematocrits in children in the Ten-State Nutrition Survey (Center for Disease Control, 1972a) may be seen from Table 4–3. The data concern families sampled in those census tracts classified in the lowest quartile for income in the state and are presented separately for states with lower per capita incomes (low income ratio states) and for those with higher per capita incomes (high income ratio states). With the exception of the 2- to 5-year-old age group in the low income ratio states, in each age-income category, anemia was least in white children and greatest in black children, with intermediate prevalence in Spanish-American children.

When classified according to income (i.e., low income ratio vs. high income ratio) and ethnic group, it is evident that age is an important factor. For example, in the Ten-State Nutrition Survey (Table 4–3) anemia was less prevalent among children between 6 and 12 years of age than among those from 2 through 5 years of age.

Children More Than 10 Years of Age and Adolescents

As may be seen from Figure 4–1 values for hemoglobin concentration corresponding to the mean minus 2 standard deviations generally fall at or slightly below 12.0 g/100 ml for males between 10 and 14 years of age and for females between 10 and 18 years of age. The corresponding values for males more than 14 years of age are somewhat higher.

TABLE 4–3

SUMMARY OF HEMOGLOBIN CONCENTRATIONS LESS THAN 11.0 gm/100 ml AND HEMATOCRITS LESS THAN 34 IN CHILDREN EXAMINED IN TEN-STATE NUTRITION SURVEY
(Center for Disease Control, 1972a)

	Low Income Ratio States*				High Income Ratio States*			
	Hemoglobin		Hematocrit		Hemoglobin		Hematocrit	
Age†	Number	Percent <11.0 gm/100 ml	Number	Percent <34	Number	Percent <11.0 gm/100 ml	Number	Percent <34
2–5 years								
White	313	13.1	328	11.8	845	7.9	1056	10.6
Black	1060	34.0	1188	29.6	305	19.0	382	15.3
S.A.**	158	9.5	157	14.1	330	16.0	332	11.1
6–12 years								
White	876	4.5	869	3.1	2650	1.3	2710	2.3
Black	2435	16.2	2443	12.6	996	9.1	993	4.2
S.A.	572	5.2	566	4.7	832	4.4	829	1.7
13–16 years								
Males								
White	197	1.5	195	1.0	539	0.0	539	0.2
Black	545	4.1	539	3.0	206	2.5	200	0.5
S.A.	126	4.8	126	4.8	151	1.4	149	0.7
Females								
White	193	3.6	193	1.0	507	0.6	514	1.0
Black	628	12.1	629	6.2	221	14.0	216	7.4
S.A.	156	5.1	156	3.2	167	7.2	167	1.8

*Low income ratio states include Kentucky, Louisiana, South Carolina, Texas and West Virginia; high income states include California, Massachusetts, Michigan, New York and Washington.

†Age 2–5 years indicates that child has reached second birthday and has not yet reached fifth birthday.

**Spanish-American.

Among adolescents from 12 to 18 years of age in the Health and Nutrition Examination Survey (Abraham et al., 1974), only 1.9% of males and 3.7% of females demonstrated hemoglobin concentrations less than 12.0 g/100 ml.

Results of the Ten-State Nutrition Survey (Center for Disease Control, 1972a) demonstrate that prevalence of hemoglobin concentrations less than 12.0 g/100 ml is generally greater among low income than among higher income groups (Table 4–4). Black children and adolescents demonstrate greater prevalence of low hemoglobin concentrations than do white individuals of the same age and sex.

Iron Requirements

To remain in satisfactory iron nutritional status, the child must absorb sufficient iron to provide amounts necessitated by growth and to replace losses that occur through the gastrointestinal tract, through the skin and in the urine. Girls who have reached the menarche must, in addition, absorb enough iron to replace losses that occur through menstruation. Requirements for absorbed iron and dietary requirements for iron will be considered separately.

TABLE 4–4

PERCENT OF ADOLESCENTS 13 TO 17 YEARS OF AGE WITH CONCENTRATIONS OF HEMOGLOBIN LESS THAN 12.0 gm/100 ml. TEN-STATE NUTRITION SURVEY (Center for Disease Control, 1972a)

	Males		Females	
	Number	*Hb < 12.0 gm/100 ml (%)*	*Number*	*Hb < 12.0 gm/100 ml (%)*
Low Income Ratio States				
White	197	5.0	193	16.0
Black	543	19.0	628	41.2
Spanish American	126	11.2	156	14.1
High Income Ratio States				
White	539	2.0	507	6.7
Black	206	16.1	221	37.1
Spanish American	151	6.7	167	22.8

Requirements for Absorbed Iron

We have previously estimated requirements for absorbed iron to be approximately 0.7 mg/day during the first year of life and 0.8 mg/day between 1 and 3 years of age (Fomon, 1974a). These estimates were (and still are) believed to be generous.

Estimates of requirements for absorbed iron by children from 3 to 19 years of age are presented in Table 4–5. For each age interval and for separate sexes beginning at age 10 years, a crude estimate of yearly gain in fat-free body mass during the age interval is given. An estimate of body weight at the end of the age interval is also given. The requirement for absorbed iron for growth was calculated on the assumption that 74 mg of iron are included in each kilogram of newly gained fat-free body mass—i.e., the iron content of fat-free body mass of the adult (Widdowson and Spray, 1951). Nonmenstrual losses of iron (gastrointestinal, dermal and urinary) amount to approximately 0.86 mg/day in the adult (Green et al., 1968) and for the child over 3 years of age are assumed to be 0.012 mg/kg body weight. Our calculations have utilized the weight applicable at the end of the age interval. Menstrual losses have been assumed to be 0.5 mg/day, a value equivalent to loss of 14 mg of iron or 4.1 g of hemoglobin every 28 days. This amount corresponds with loss of approximately 34 ml of blood with hemoglobin concentration of 12 g/100 ml. The 50th and 75th percentile values for menstrual blood loss by 15-year-old girls were reported by Hallberg et al. (1966) to be 28.4 and 44.5 ml, respectively.

It is evident that the requirements for absorbed iron may be greater than those indicated in Table 4–5 in the case of girls with regular menses occurring before age 12 years. In addition, greater amounts of absorbed iron may be required by girls with menstrual losses substantially greater than 34 ml per 28 days. However, it should be noted that many of the 12- to 15-year-old girls who have regular menstrual periods will be well beyond the peak of the adolescent growth spurt and will be gaining considerably less than the assumed 3.5 kg of fat-free body mass per year. Requirements for iron for growth will therefore be considerably reduced.

Dietary requirements

Assuming that 10% of dietary iron is absorbed (p. 108), the requirements for iron average 10 times the values for total requirements for absorbed iron. These values are summarized in Table 4–6. That such estimates are somewhat generous seems likely because the percentage of children and adolescents with intakes below the estimated requirements (p. 112) is so much greater than the percentage with hemoglobin concentrations below the normal range.

TABLE 4-5

ESTIMATED REQUIREMENTS FOR ABSORBED IRON

Age* (years)		Estimated Gain FFBM (kg/year)	Estimated Body Weight at End of Age Interval (kg)	Requirements for Absorbed Iron (mg/day)			
				Growth	*Losses*		*Total*
					Non-Menstrual	*Menstrual*	
3–6	M+F	2.0	21	0.4	0.3	–	0.7
6–10	M+F	2.5	32	0.5	0.4	–	0.9
10–12	M	3.8	40	0.8	0.5	–	1.3
	F	4.5	42	0.9	0.5	–	1.4
12–15	M	5.5	57	1.1	0.7	–	1.8
	F	3.5	54	0.7	0.6	0.5	1.8
15–17	M	4.0	67	0.8	0.8	–	1.6
	F	0.5	56	0.1	0.7	0.5	1.3
17–19	M	1.0	70	0.2	0.8	–	1.0
	F	–	57	–	0.7	0.5	1.2

*Age 3–6 years indicates that a child has reached the third birthday and has not yet reached the sixth birthday.

TABLE 4–6

DIETARY REQUIREMENT FOR IRON (mg/day)

Age* (years)	Males	Females
0– 1	7	7
1– 3	8	8
3– 6	7	7
6–10	9	9
10–12	13	14
12–15	18	18
15–17	16	13
17–19	10	12

*Age 1–3 years indicates that a child has reached the first birthday and has not yet reached the third birthday.

Absorption of Iron

A mixed diet always provides some iron contained within plant or animal tissue and usually includes various iron compounds used to supplement the iron content of certain foods. Cooking utensils and water contribute small amounts of iron. In addition, medicinal iron (drops or tablets) contributes to total intake of iron by some individuals. Finally, although dietary iron is absorbed mainly in the duodenum and upper jejunum, cells sloughed from the proximal intestinal mucosa may be digested and provide a relatively small and somewhat variable contribution to the total amount of absorbed iron.

It may be concluded from evidence cited in various reviews (e.g., Bothwell and Charlton, 1970; Dagg et al., 1971; Levitan and Wilson, 1974) that amounts of iron absorbed by individuals consuming identical diets will be influenced by such factors as age, sex, iron status and physiologic state. The efficiency of absorption is determined by the size of body iron stores and by a number of factors (e.g., residence at high altitude) that influence erythropoietic activity.

Absorption of Iron Naturally Present in Food

Iron from vegetable sources is generally less well absorbed than that from animal sources (Fig. 4–3). Of the animal sources, heme iron (from

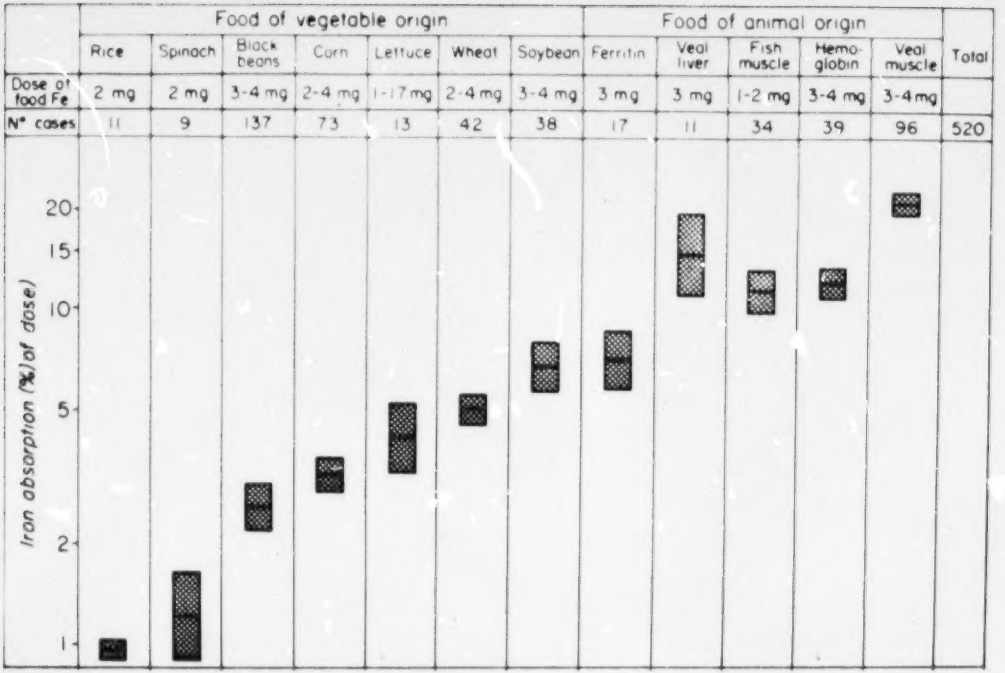

Figure 4–3 Iron absorption from various plant and animal foods. Each horizontal line indicates the geometric mean and the cross-hatched area shows the limits of one standard error (Layrisse and Martinez-Torres, 1971).

meat, fish, etc.) appears to be more readily absorbed than non-heme iron (from eggs, dairy products). Most studies (Moore and Dubach, 1951; Chodos et al., 1957; Ministry of Health, 1968; Callender et al., 1970) indicate that absorption of iron from egg by human adults is particularly poor, and the inclusion of eggs in the diet appears to interfere with absorption of iron from other sources (Chodos et al., 1957; Elwood et al., 1968).

Because heme iron is so much more readily absorbed than is non-heme iron, it may be reasonable to consider that food iron is absorbed from two pools—a heme iron pool and a non-heme iron pool (Martinez-Torres et al., 1974; Björn-Rasmussen et al., 1974). Although a two-pool model is almost certainly an oversimplification, such a model at least seems more reasonable than a single pool model.

From study of 74 adult subjects given 2–4 mg of iron (mainly heme iron) in the form of veal liver, Martinez-Torres et al. (1974) demonstrated approximately 11% absorption in normal adults, 20% in those with moderate reduction in iron stores and 30% in those with marked reduction in iron stores. The authors point out that these results are similar to results reported by other investigators with chicken liver and rabbit liver and are also similar to their own previous studies with veal muscle. When non-heme iron from corn was included in the same meal as veal liver, the absorption of iron from corn was markedly improved while the absorption of heme iron was decreased.

Healthy young men in excellent hematologic status were studied (Björn-Rasmussen et al., 1974) while receiving a diet similar in heme and non-heme iron content to that consumed in Swedish military service. With an intake in one day of 16.4 mg of non-heme iron and 1.0 mg of heme iron, absorption averaged 7.2% (5.3% of the non-heme iron and 37.3% of the heme iron). One may presume that percentage absorption from the same diet might be somewhat greater for children than for adults, especially if hematologic status were less satisfactory. In addition, percentage absorption would be expected to be greater if a larger percentage of the total iron intake were in the form of heme iron.

Absorption of Iron Added to Foods

Standards of identity for iron enrichment of wheat flour, bread and other flour-containing baked products were first published in the Federal Register in 1941. Since that time other cereal foods have been added to the original list of iron-enriched foods. Waddell (1973, 1974) has reviewed the early studies relating to the bioavailability of iron compounds added to foods. Ferrous sulfate, ferric orthophosphate, sodium iron pyrophosphate and so-called "reduced iron" comprise the major sources of iron used in enrichment.

Fritz et al. (1975) have evaluated the relative bioavailability (ferrous sulfate employed as a reference) of a large number of iron compounds and food sources when added to the diets of anemic rats and chicks. Later work (Pla et al., 1973; Shah and Belonje, 1973; Anderson et al., 1974; Motzok et al., 1975) demonstrated that the bioavailability of elemental iron powders is related to particle size. Patrick (1973) has clarified some of the nomenclature regarding various forms of elemental iron powders, pointing out that "reduced iron" has often been confused with electrolytic or carbonyl iron.

The iron salt commonly employed for fortification of commercially prepared infant formulas in the United States is ferrous sulfate. This salt appears to be well absorbed from milk-based formulas (Marsh et al., 1959; Gorten et al., 1963; Gorten and Cross, 1964; Gorten, 1965; Andelman and Sered, 1966).

Bioavailability of the forms of iron currently added to other infant foods has not been systematically investigated. Changes in recipe formulation and in sources of iron used to supplement the products marketed by various manufacturers often negate the usefulness of studies reported in the literature. For example, Rios et al. (1975) reported that less than 1% of sodium iron pyrophosphate incorporated into dry mixed cereal was absorbed. However, at least two years before the report was published, each of the three baby food manufacturers had discontinued use of sodium iron pyrophosphate in dry cereals in favor of electrolytic iron powder. Electrolytic iron of small particle size (95% passage through a 325 mesh screen) is currently used by all manufacturers, a form reported by Rios et al. (1975) to exhibit bioavailability at least as good as that of ferrous sulfate when used to supplement cereal. Ferrous sulfate is now added to most cereal with fruit combinations produced by two baby food manufacturers. The bioavailability of iron in these products can be assumed to be relatively good.

Cook et al. (1973) in a well-designed study of human adults, in which each subject served as his own control, determined the relative bioavailability of ferrous sulfate, sodium iron pyrophosphate, ferric orthophosphate and "reduced iron" baked into dinner rolls. Absorption of sodium iron pyrophosphate, ferric orthophosphate and "reduced iron" was 5, 31 and 95, respectively, of the absorption of ferrous sulfate. Less than 1% of sodium iron pyrophosphate and ferric orthophosphate was absorbed whereas 8.6% of the "reduced iron" was absorbed. Absorption of ferrous sulfate baked into the rolls ranged from 0.9 to 48.9% with absorption from rolls approximately 25% that from the same dose of iron given in the form of a solution of ferrous sulfate. When rolls supplemented with 3 mg of ferrous sulfate were consumed with meals, absorption averaged 6.3 and 2.1% from meals with and without meat protein, respectively.

Absorption of Therapeutic Doses of Iron

Brise and Hallberg (1962) compared the absorption of 14 different iron salts with that of ferrous sulfate. All of the iron compounds were given as solutions, each containing 30 mg of iron after an overnight fast with no food or water for 2 hours after the dose. All ferric compounds were less well absorbed than were ferrous compounds. Ferric versenate was found to be very poorly absorbed whereas Anderson et al. (1974) reported disodium iron-EDTA to be 90% as effective as ferrous sulfate in preventing anemia in growing miniature pigs.

Speculation

Although the weight of evidence suggests that absorption of iron by normal adult males in good iron nutritional status is less than 10% of the amount provided by a usual mixed diet, percent absorption by infants, children and adolescents is likely to be greater than that by adults. For purposes of estimating quantity of iron absorbed from the diet by individuals less than 18 years of age, it has been assumed in this chapter that absorption by children averages 10% of intake (Table 4–6).

Dietary Intake of Iron

From Table 4–7 it is evident that a high percentage of preschool children receive intakes of iron less than the estimated requirement (Table 4–6) and that such low intakes apply to all income groups. The data from the Health and Nutrition Examination Survey (Abraham et al., 1974) are similar: 60% of 1010 children between 1 and 5 years of age living in families with incomes classified as above the poverty level were found to have intakes of iron less than 8 mg/day; 62% of 448 children of the same age living in families with incomes classified as below the poverty level were found to have intakes of iron less than 8 mg/day.

Median intakes of iron by children and adolescents of various ages are presented in Table 4–8 for the Health and Nutrition Examination Survey (Abraham et al., 1974) and in Table 4–9 for the Ten-State Nutrition Survey (Center for Disease Control, 1972b). The data indicate that for most age groups, median intakes of iron are only slightly above or are actually less than the amounts recommended (Table 4–6). Thus, it is evident that iron intakes are less than the estimated requirements in a high percentage of children. The relatively high prevalence of low hemoglobin concentrations is therefore not surprising.

TABLE 4-7

SUMMARY OF DATA ON LOW INTAKES OF IRON IN NATIONAL PRESCHOOL NUTRITION SURVEY (Owen et al., 1974)*

Age† (years)	Lowest Income Group**		Other Income Group	
	Number	*Iron Intake <8 mg/day (% of children)*	*Number*	*Iron Intake <8 mg/day (% of children)*
1–2	120	68	512	63
2–3	114	72	550	60
3–4	132	60	586	52
4–5	120	50	563	54
5–6	113	42	578	37

*The actual values presented do not appear as such in the survey report but have been received as a personal communication from Owen (1976).

†Age 1–2 years indicates that a child has reached the first birthday and has not yet reached the second birthday.

**Accounted for lowest 18% of families by Warner classification.

The ratio of iron to energy content of the diets of children in the Health and Nutrition Examination Survey demonstrated median values of approximately 5 mg of iron per 1000 kcal for children 1 through 5 years of age and 4.8 to 5.5 mg per 1000 kcal for the two sexes and various ethnic groups of children 6 through 17 years of age.

Data from the Ten-State Nutrition Survey concerning 1,351 male and 1,460 female teenagers suggested that approximately 23% of calories were obtained from foods consumed between meals. The iron content of foods consumed between meals was approximately 3.4 mg per 1000 kcal whereas the iron content of food consumed at mealtime was approximately 5.9 mg per 1000 kcal (Thomas and Call, 1973).

Means of Achieving Dietary Requirements

In our previous considerations of infants and children less than 3 years of age (Fomon, 1974), we have estimated the requirement for a nutrient and have then added a safety factor to reach an "advisable intake." In the case of such nutrients as protein (Fomon, 1974b) and calcium (Fomon, 1974c), the advisable intake has been set at a value only 20% above the estimated requirement. In such instances we had relatively high confidence in our estimates and little reason to suspect wide variability in requirement in the same healthy subject from one

TABLE 4-8

MEDIAN INTAKES OF IRON IN RELATION TO AGE, RACE AND INCOME IN HEALTH AND NUTRITION EXAMINATION SURVEY (Abraham et al., 1974)

Age* (years)	Income**	White		Black	
		Number	*Iron Intake (mg)*	*Number*	*Iron Intake (mg)*
1- 6	Low	180	7.0	265	7.0
	Higher	791	7.1	208	6.9
6-12	Low	133	10.3	177	8.2
	Higher	587	9.7	131	10.0
12-18	Low	93	9.7	168	9.7
	Higher	582	11.3	143	10.5

*Age 1-6 years indicates that a child has reached the first birthday and has not yet reached the sixth birthday.

**"Low" refers to families with incomes below the poverty index ratio (Orshansky, 1965; Current Population Reports, 1969) and "higher" refers to families with incomes above that ratio.

TABLE 4-9

IRON INTAKES LESS THAN 13 mg/day BY INDIVIDUALS 10 TO 17 YEARS OF AGE IN TEN-STATE NUTRITION SURVEY (Center for Disease Control, 1972b)

Age** (years)	Low Income Ratio States*		High Income Ratio States*	
	Number	*<13.0 mg/day (% of individuals)*	*Number*	*<13.0 mg/day (% of individuals)*
10-12				
Males	359	65	473	54
Females	557	75	739	70
12-15				
Males	706	61	918	48
Females	819	73	897	68
15-17				
Males	258	49	289	42
Females	317	78	294	65

*See footnote to Table 4-2.

**The designation 10-12 years refers to individuals who have reached the 10th birthday and not yet reached the 12th birthday.

time to another, or between healthy subjects in the same age group. In the case of sodium (Fomon, 1974c), on the other hand, it was evident that a large element of uncertainty in the requirement resulted from poorly documented and probably widely variable dermal and urinary losses. Thus, the advisable intake value was set at three times the estimated requirement.

In our consideration of advisable intakes of iron by infants and children less than 3 years of age (Fomon, 1974a), we believed that our estimates of requirements were so generous that the advisable intake could be set, tentatively, at the same value. With some exceptions (e.g., girls who have established regular menstrual periods with average or above average menstrual losses of blood before completion of the growth spurt), we consider our estimates for iron requirements for older children to be generous also. The following discussion concerns means of achieving intakes of iron equal to the estimated requirements.

Infants

During the first year of life intakes of iron of 7 mg daily will rarely be achieved unless the infant receives an iron-fortified formula, an iron-fortified infant cereal or medicinal iron.

During the early months of life, breastfed infants should receive a supplement of iron sufficient to provide 7 mg daily in the form of ferrous sulfate or other preparation of similar bioavailability. Older breastfed infants should receive either an iron supplement or an iron-fortified infant cereal.

Precooked, iron-fortified, dry infant cereals are commonly combined with 6 parts by weight of milk or formula for infant feeding (Anderson and Fomon, 1971). Thus, one-half ounce of dry cereal (approximately 15 gm), containing 6.5 mg of elemental iron, will make a serving of 100 gm of cereal as fed. Although this is a larger serving than might be suitable before 6 months of age, those between 6 and 18 months of age may conveniently receive 6.5 mg of iron in this manner. Total iron intake will then almost certainly meet the requirement.

Infants fed evaporated milk formulas should receive additional iron as described for breastfed infants.

Infants fed commercially prepared formulas should receive those fortified with iron. Other sources of iron are not required. Older infants fed fresh milk should regularly receive an iron-fortified infant cereal or an iron supplement. The practice of discontinuing feeding of infant cereals at about 6 months of age should be corrected. *Parents should be taught that dry, precooked, iron-fortified infant cereals are a good source of iron which should be fed until 18 months of age.*

Premature infants should also receive approximately 7 mg of iron daily. Because supplemental iron may enhance red cell hemolysis in premature infants, particularly in those not receiving adequate supple-

ments of vitamin E (Melhorn and Gross, 1971), some neonatologists defer iron supplementation until 2 or 3 months of age. Whether it is better to start iron supplementation at 2 or 3 months of age or within a few weeks of birth is unresolved (Dallman, 1974). It should be understood that, if iron supplementation is deferred until 2 or 3 months of age, the requirement for iron will be proportionately greater thereafter, perhaps 9 mg/day. It seems preferable, however, to start iron supplementation soon after birth and to provide adequate amounts of vitamin E. A water-soluble vitamin E preparation, d-α-tocopheryl polyethylene glycol 1,000 succinate, is better absorbed than is the fat-soluble form, d-α-tocopheryl acetate, that is commonly used (Gross and Melhorn, 1974). Closer medical supervision is desirable for premature than for full-term infants. Hemoglobin concentration or hematocrit should be determined at least monthly during the first few months after discharge from the hospital and subsequently at less frequent intervals.

Preschool Children

It seems likely that most children between 2 and 5 years of age receive 4 to 6 mg of iron per 1000 kcal. Because energy intakes will generally range between 1100 and 1500 kcal, intakes of iron will probably range from 4 to 9 mg daily. Many children in this age group will receive quite low intakes of iron, perhaps 4 to 6 mg daily, and will be likely to be in unsatisfactory iron nutritional status. Such low intakes are particularly likely in children who obtain a relatively large fraction of their energy intake from milk.

It is evident that in many cases the diet can be modified to provide larger amounts of iron through the simple measure of placing moderate restrictions on intakes of foods low in iron content that commonly contribute a substantial percentage of energy intake. Notable among these, in addition to milk, are other dairy products, bakery goods unfortified with iron, candies and soft drinks. When diets of preschool children provide 5.5 mg of iron or more per 1000 kcal, iron intakes are likely to be adequate.

Intake of milk should generally not exceed 24 oz (720 ml) daily. By use of skim or 2% milk for the majority of children over 2 years of age, the energy intake from this poor source of iron can be decreased without interfering with intake of other essential nutrients provided by milk. As in the case of infants, consumption of bakery goods unfortified with iron and of candies and soft drinks should be curtailed—a step that will also be beneficial in prevention of dental caries.

Although it is obvious that adequate intakes of iron can be provided by use of iron supplements, it seems preferable to educate parents and children to more acceptable dietary selection rather than to deal with the problem through supplements.

School-age Children

In general, intakes of iron by children from 5 to 10 years of age more nearly meet the requirements than do those of any other age group during childhood. The major exception is in the lowest income group, especially among blacks. Dietary changes such as those described for preschool children should be encouraged among school-age children as well.

Males 10 to 19 Years of Age

As may be seen from Table 4–6, estimated requirements for iron for male adolescents are greatest between 12 and 15 years of age. The 50th percentile value for calorie intakes by males 12 to 15 years of age in the high income ratio states in the Ten-State Nutrition Survey was approximately 2500 kcal/day. Usual American diets provide slightly less than 6 mg of iron per 1000 kcal. Thus, 2500 kcal may be expected to provide about 15 mg of iron—an intake less than the estimated 18 mg of dietary iron required. Unless there is a change in the national diet as would come about through a greater level of iron fortification of bread, one must assume that the iron requirements of many adolescent males 12 to 15 years of age will not be met from food sources. Educational efforts aimed at awareness of the low iron content of certain foods and the relatively high iron content of other foods are desirable. In some instances, administration of medicinal iron supplements will be warranted.

Among males 15 to 17 years of age the 50th percentile energy intake is between 2750 and 3000 kcal/day. If the diet provides 6 mg of iron per 1000 kcal, an intake of 2750 kcal will provide 16.5 mg of iron—an amount slightly greater than the estimated 16 mg requirement value. Thus, supplements of iron are probably not required for most males in this age group.

In view of the lesser requirement for iron after the adolescent growth spurt, males 18 and 19 years of age are likely to have little difficulty in achieving adequate intakes of iron from commonly consumed diets.

Females 10 to 19 Years of Age

Because the iron content per unit of energy is similar in diets consumed by males and females and because energy intakes of females are less than those of males, difficulties in achieving adequate intakes of iron from dietary sources are greater for females than for males, at least in the age ranges 10 to 17 years. In fact, between 10 and 12 years of age, the estimated iron requirement is actually greater for females than for males (Table 4–6).

The 50th percentile intake of energy by females 10 to 12 years of age in the Ten-State Nutrition Survey was between 1750 and 2000 kcal

per day in high income ratio states. It is evident that a diet providing 6 mg of iron per 1000 kcal will generally not provide the estimated requirement (14 mg) in this energy intake. With only modestly increased intakes and considerably increased needs for iron between 12 and 15 years of age, diet alone is unlikely to provide adequate intakes of iron in this age group either. Iron supplements will therefore be desirable for most females 12 to 17 years of age. After the 17th birthday, the great majority of women will be able to achieve adequate intakes of iron from the diet if they are cautious to avoid consuming a substantial proportion of their energy needs in the form of foods with a low ratio of iron to energy.

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